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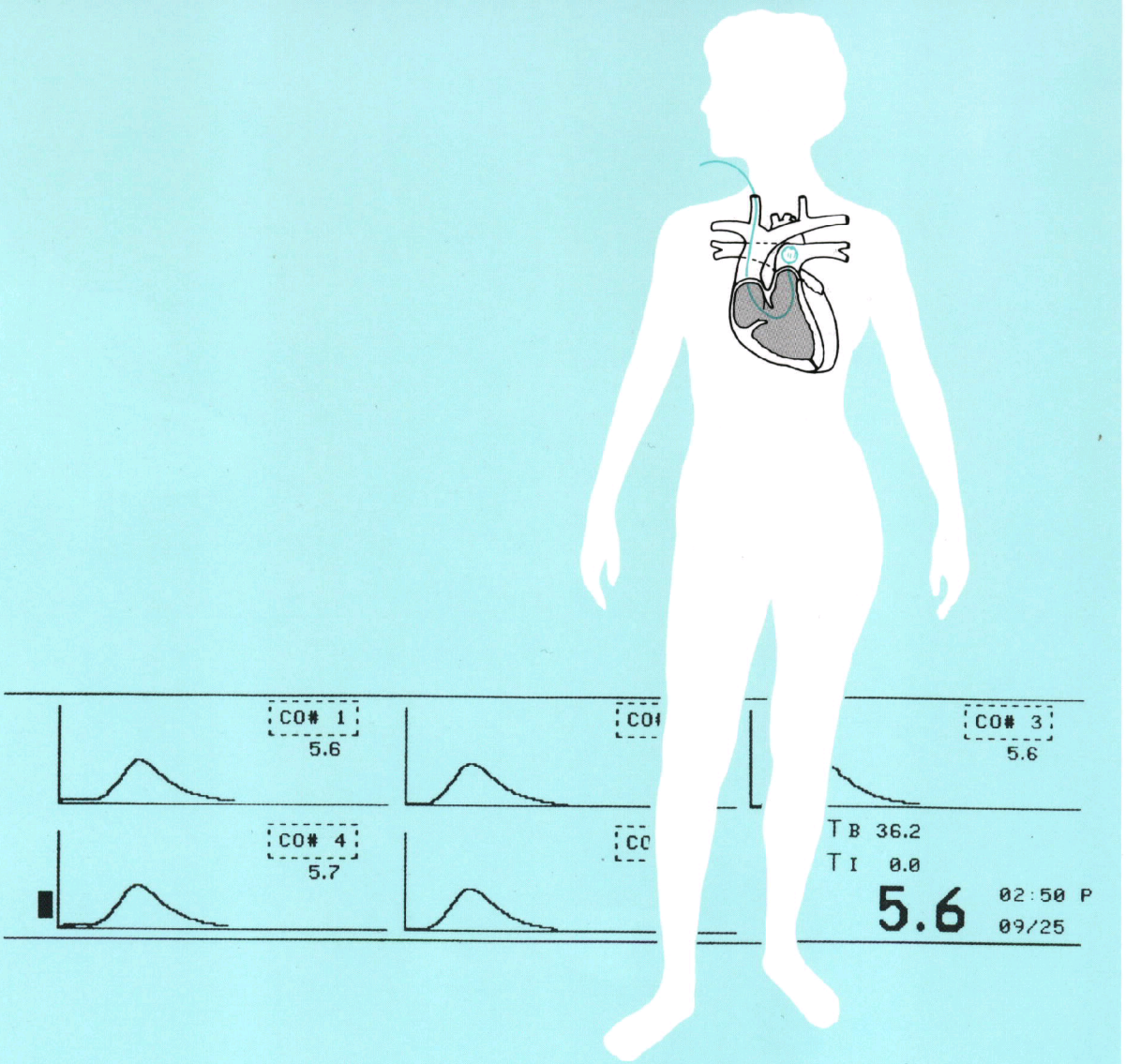
BIOPHYSICAL MEASUREMENT SERIES

CARDIAC OUTPUT



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CARDIAC OUTPUT

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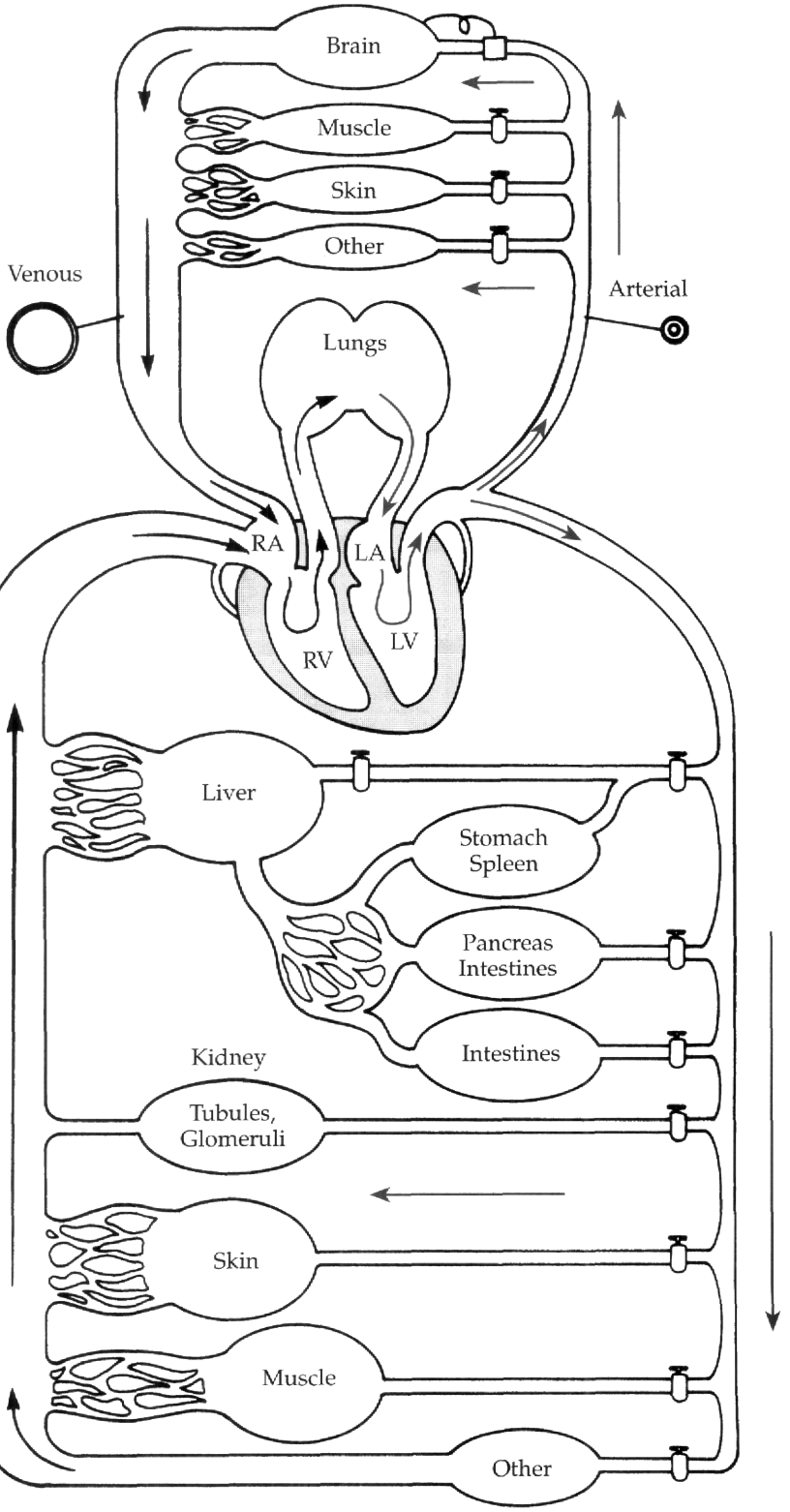
INTRODUCTION

This publication describes techniques for measurement of cardiac output, with emphasis on thermodilution methodology. The concept of measuring cardiac output in humans is attributed to Adolph Fick who, in 1870, postulated a technique that bears his name. For the next 100 years, cardiac output was measured by a variety of techniques, but only in specialized laboratory settings. Not until the introduction of the Swan-Ganz pulmonary artery catheter in 1971 did thermodilution cardiac output measurement become widely available for routine patient care. Perhaps more than any other technological development, the Swan-Ganz pulmonary artery catheter with thermodilution cardiac output capability catalyzed the beginning of modern critical care medicine.

Figure 1.1 — The heart acts as a mechanical pump that provides the energy for the flow of blood.



1.0 HISTORY AND OVERVIEW OF CARDIAC OUTPUT MEASUREMENT IN HUMANS

1.1 *Cardiac Output, Basic Concepts*

Cardiac output is the volume of blood pumped by the heart per unit time, usually expressed in liters per minute (l/min).[1] Blood flow is not constant, however. The heart operates as a pulsatile pump that ejects a bolus of blood, known as the stroke volume, with each cycle of contraction. Cardiac output (CO) is the stroke volume (SV) times heart rate (HR),

$$CO = SV \times HR$$

where
CO = cardiac output (l/min)
SV = stroke volume, liters
HR = heart rate (per minute).

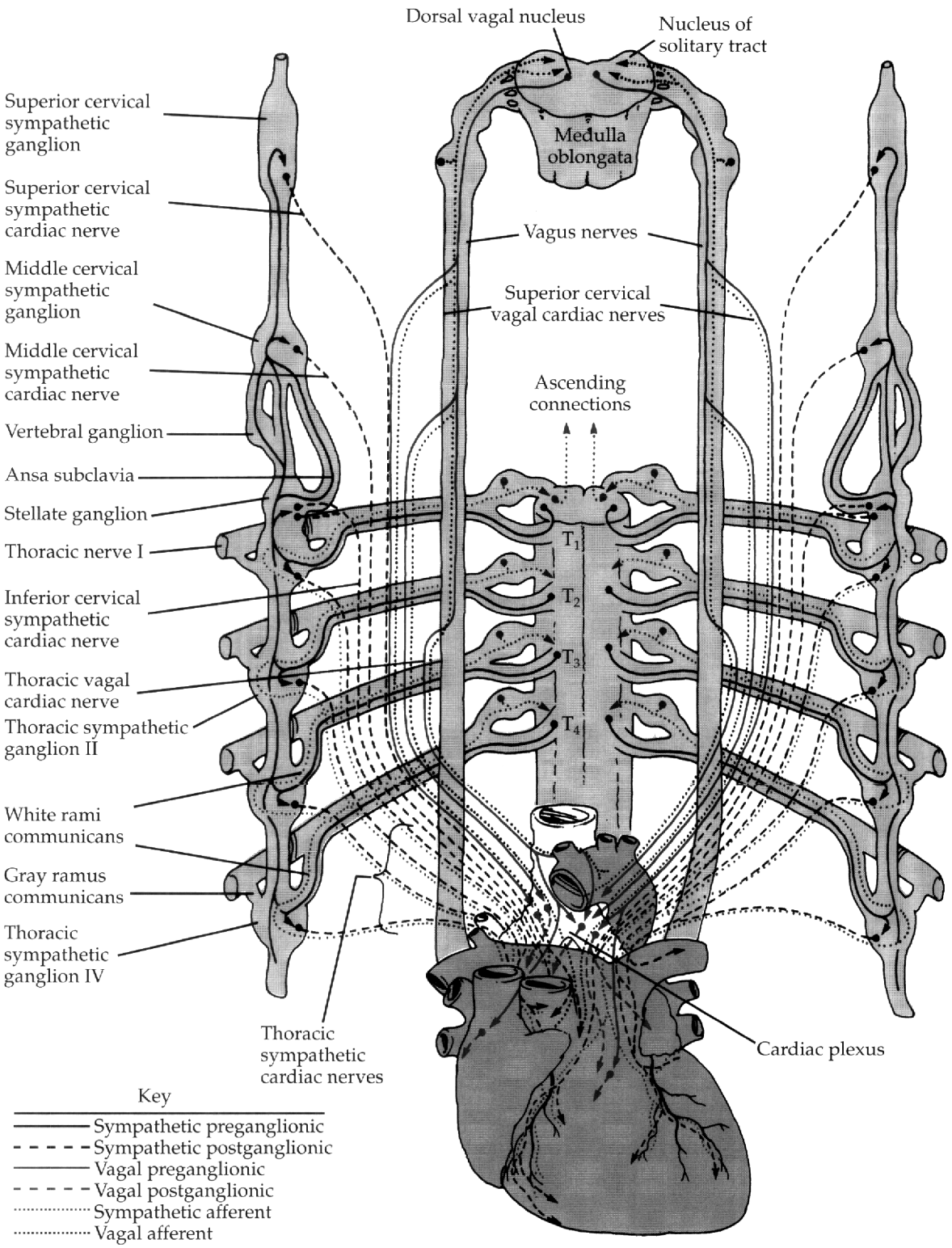
A complex set of interrelated physiological variables determine the magnitude of cardiac output, including the volume of blood in the heart (preload), the downstream resistance to ejecting blood from the heart (afterload), and the contractility of the heart muscle (Figure 1.1).

The metabolic requirements of the body also influence cardiac output. The integration of the heart and metabolic demand is facilitated by a complex network of nerves, the autonomic nervous system, that regulates the activity of the cardiovascular system (Figure 1.2).[2] A variety of hormones also have important cardiovascular effects.

The regulation of cardiac output is a complex affair. A single measurement of cardiac output represents the net effects of many interacting physiological systems. Cardiac output reflects not only the functional state of the heart but also the response of the entire circulatory system to physiological demands, including acute and chronic disease and the impact of therapeutic interventions.[3]

Basal cardiac output is related to body size and varies from approximately 4 to 7 l/min in adults. To normalize the cardiac output for body size, the cardiac output may be divided by the body surface area, giving the cardiac index (CI):

Figure 1.2 — The pumping function of the heart is regulated by a complex system of nerves known as the autonomic nervous system.



$$CI = \frac{CO}{BSA}$$

where CI = cardiac index (normally 2.5 to 3.5 l/min/m^2)
CO = cardiac output (l/min)
BSA = body surface area (in square meters) obtained from a nomogram based on height and weight.

1.2 *Fick Technique*

The first technique for measuring cardiac output in humans was described by Adolph Fick in 1870.[4] Fick postulated (but never actually made the measurement himself) that cardiac output could be calculated from the difference in oxygen content between the mixed venous (pulmonary artery) and arterial blood and the total body oxygen consumption (Figure 1.3):[5,6]

$$CO = \frac{O_2 \text{ consumption}}{(A\text{–}V\ O_2\Delta)}$$

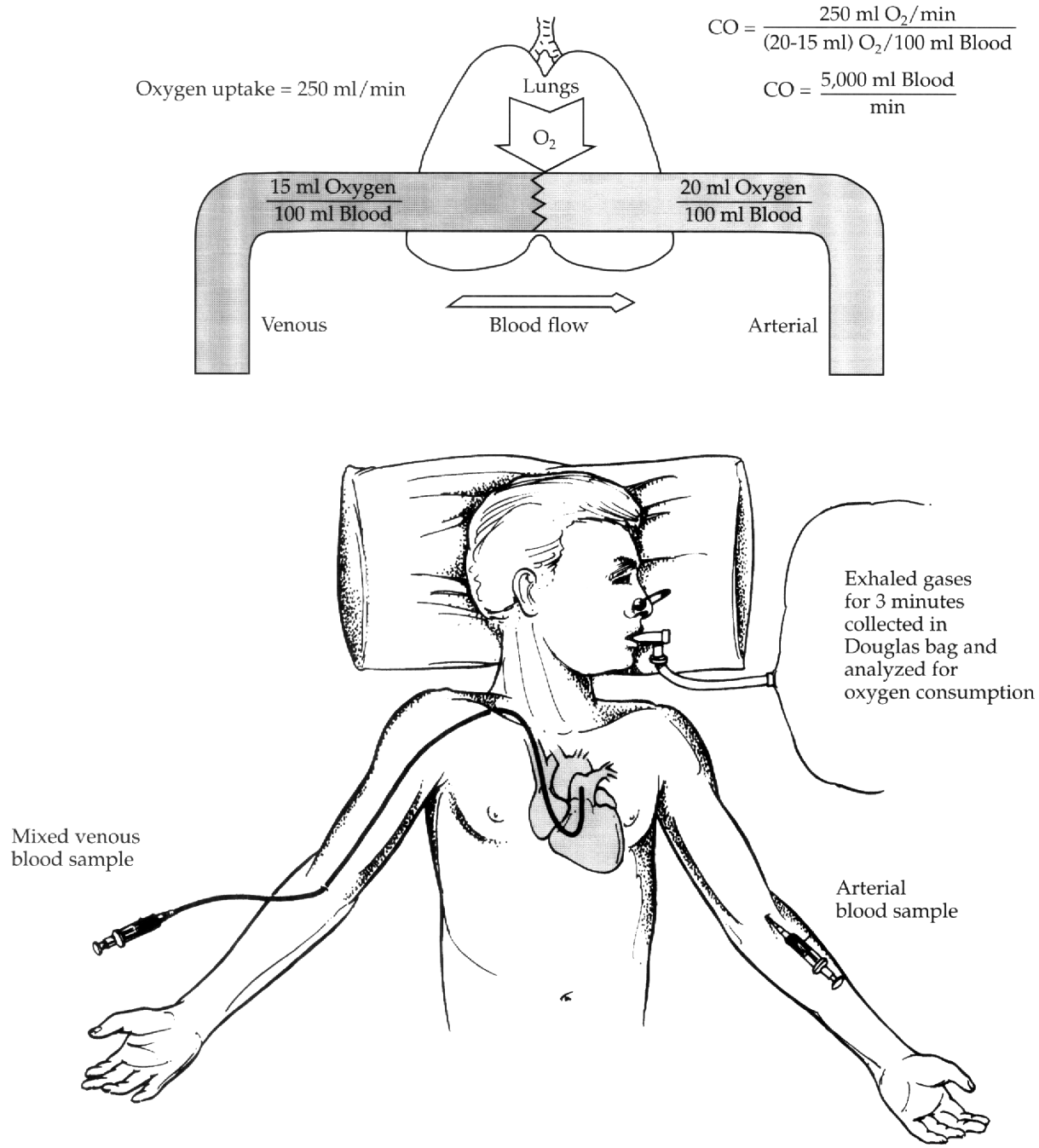
where CO = cardiac output
A-V $O_2\Delta$ = difference in oxygen content between mixed venous and arterial blood [(20-15) ml O_2/100 ml blood]
O_2 consumption = total body oxygen utilization (ml/min).

Oxygen content of venous or arterial blood is calculated as:

$$O_2 \text{ content} = Hb \times \%sat \times 1.34 \text{ ml } O_2/g\ Hb + (0.003 \times PO_2)$$

where Hb = hemoglobin concentration (g/dl)
%sat = % saturation, the % of hemoglobin that is carrying oxygen
1.34 ml O_2/g Hb = maximum oxygen carrying capacity of hemoglobin at 100% saturation
0.003 = volume of dissolved oxygen per torr PO_2 (ml/torr)
PO_2 = partial pressure of oxygen (torr).

Figure 1.3 — The Fick method for determining cardiac output: Venous blood passes through the lungs, takes up oxygen, and returns to the body through the arterial circulation. By measuring the rate of oxygen uptake and the oxygen content of venous and arterial blood, the cardiac output can be calculated. Performing these measurements requires an arterial and central venous catheter and a closed breathing circuit for the measurement of oxygen consumption.



Hemoglobin saturation is measured with a co-oximeter. Partial pressure of arterial oxygen (P_aO_2) and partial pressure of mixed venous oxygen (P_vO_2) are measured with a blood-gas analyzer. Dissolved oxygen is a negligible part of the total oxygen content, except when the hemoglobin concentration is extremely low.

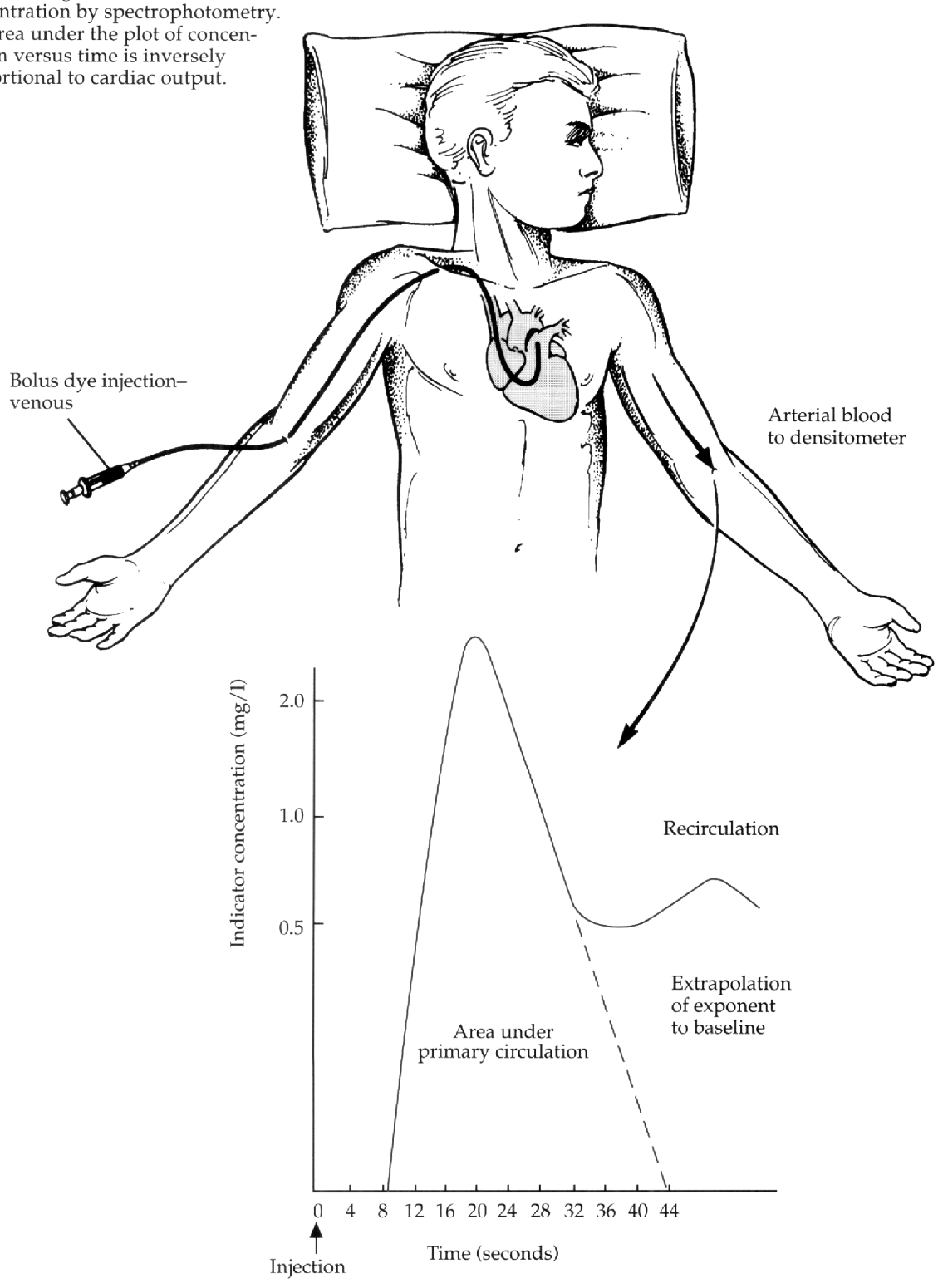
Oxygen consumption is determined by having the patient breathe into a circuit that allows the collection of all exhaled gas into a large bag. After about 3 minutes, the total volume of exhaled gas and the volume of oxygen in the bag are measured. The difference between the volume of oxygen in the exhaled gas and the volume of oxygen in a like volume of the inhaled gas (corrected to standard temperature and pressure) is the volume of oxygen consumed by the person, ordinarily about 250 ml/min for a 70 kilogram (kg) individual at rest.

The Fick method for measuring cardiac output is considered the gold standard for the physiology laboratory. Severe limitations exist for the use of this technique in the clinical setting, however. Frequent measurements are impossible because of the time needed to collect gas for the determination of oxygen consumption. Cardiac output must remain constant during the period of gas collection for reliable results. The Fick method is most accurate when cardiac output is normal or reduced. High cardiac outputs are difficult to measure accurately because the A-V O_2 difference is small and minor errors in the determination of arterial and venous oxygen content result in large errors in cardiac output.[7]

1.3 *Indicator Dilution Technique*

The indicator dilution technique (sometimes designated as the indirect Fick method) for determination of cardiac output was introduced by Stewart in 1897[8] and modified by Hamilton in 1932.[9] A measurable substance is injected into the circulation and the concentration of this substance is measured downstream from the injection site. The indicator mixes with blood and is thereby diluted. The extent of dilution, determined by measuring the downstream concentration, is inversely proportional to the blood flow.[10] A variety of indicators have been used with this technique, including inert dyes, gases, hypertonic saline and cold saline or dextrose solution (thermodilution).[6]

Figure 1.4 — The measurement of cardiac output by dye dilution requires a central venous and an arterial catheter. A bolus of dye, usually indocyanine green, is injected rapidly into the central venous circulation. Blood is withdrawn continuously from the artery and passes through a densitometer, which determines the concentration by spectrophotometry. The area under the plot of concentration versus time is inversely proportional to cardiac output.



1.4 *Dye Dilution*

Dye dilution was a popular method for determination of cardiac output in physiology and cardiac catheterization laboratories prior to the use of thermodilution. In this method, a bolus of dye, usually indocyanine green, is injected rapidly into the venous circulation near the right atrium and the downstream concentration of dye is measured from a peripheral artery. A blood sample is withdrawn continuously from the artery and passed through a densitometer that determines the concentration by spectrophotometry (Figure 1.4). A chart recorder produces a dye concentration versus time curve. The area under the curve is inversely proportional to cardiac output. The mathematical basis for the calculation of cardiac output from indicator dilution techniques is developed in Section 2.0.

Like the Fick technique, dye dilution is not suitable for routine clinical use. Calibration of the dye densitometer is difficult, and repeated determinations of cardiac output are limited by the accumulation of dye in the circulation.[11]

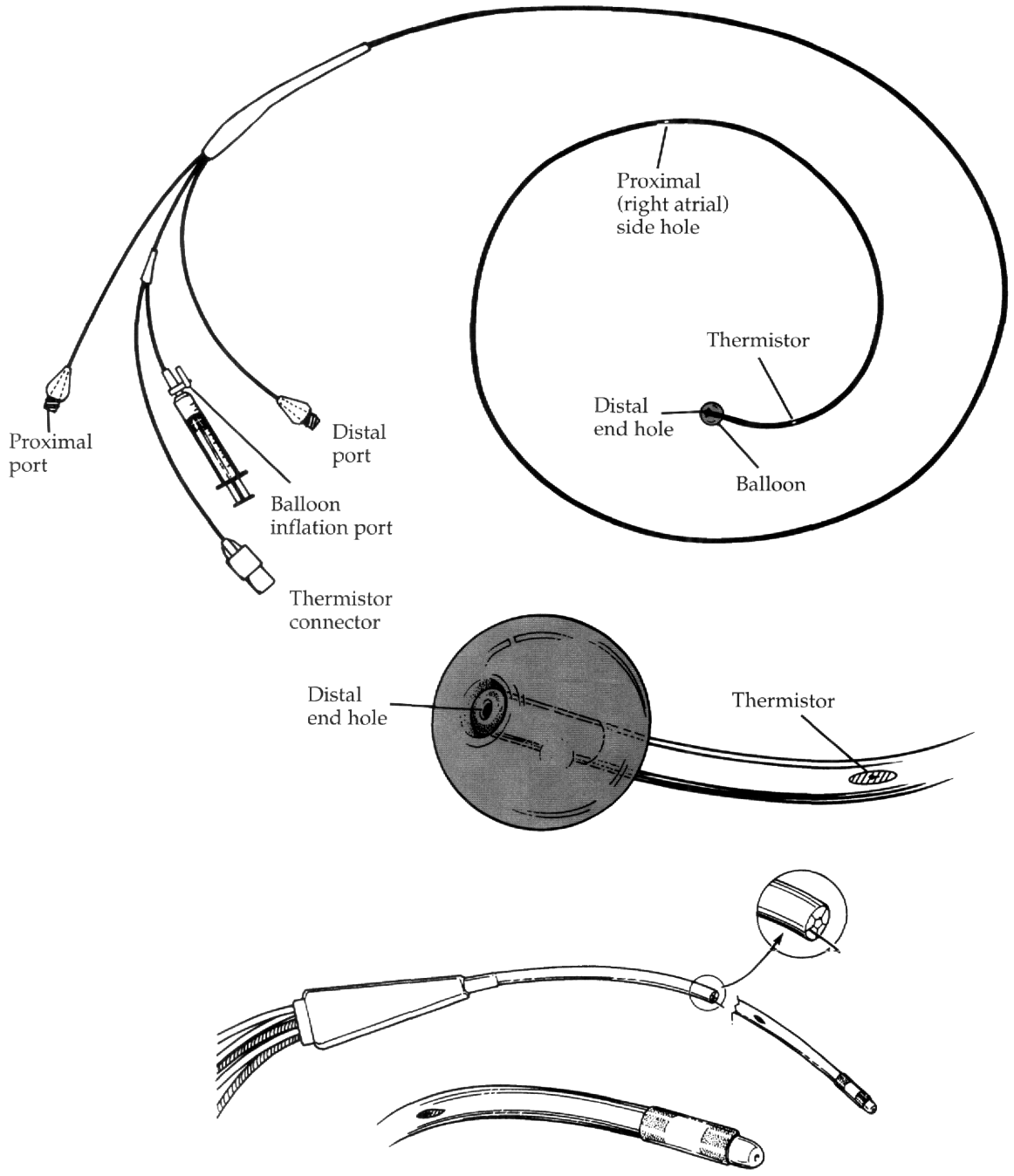
1.5 *The Swan-Ganz Pulmonary Artery Catheter and the Thermodilution Technique*

Catheterization of the human heart began in 1929 when Forssman inserted a catheter into his own right atrium. Table 1.1 provides the significant events of the history of cardiac catheterization. The introduction of the Swan-Ganz balloon-tipped, flow directed catheter in 1971 has taken right heart catheterization and cardiac output determination out of the laboratory and into the intensive care unit and operating room.[12, 13]

The Swan-Ganz pulmonary artery catheter is a multilumen catheter typically measuring 7 to 8 on the French scale (approximately 2 mm diameter) by 110 cm in length (pediatric sizes are smaller); see Figure 1.5. Near the tip is a thin-walled latex balloon that can be inflated by injection of gas (usually air) into the lumen of the catheter that is connected with the balloon.

The inflatable balloon serves two important functions: (1) to allow the catheter to be advanced into the pulmonary artery without using fluoroscopy, by floating the tip of the catheter along with the flow of blood through the right atrium, tricuspid valve, right ventricle, and pulmonic valve into the pulmonary artery; (2) once the

Figure 1.5 — A typical pulmonary artery catheter: The balloon adheres tightly to the wall of the catheter until it is inflated by the addition of approximately 1.5 ml of air. A cross-section of the catheter shows the channels for pressure monitoring (pulmonary artery and right atrium), for inflation of the balloon, and for the wire to the thermistor.



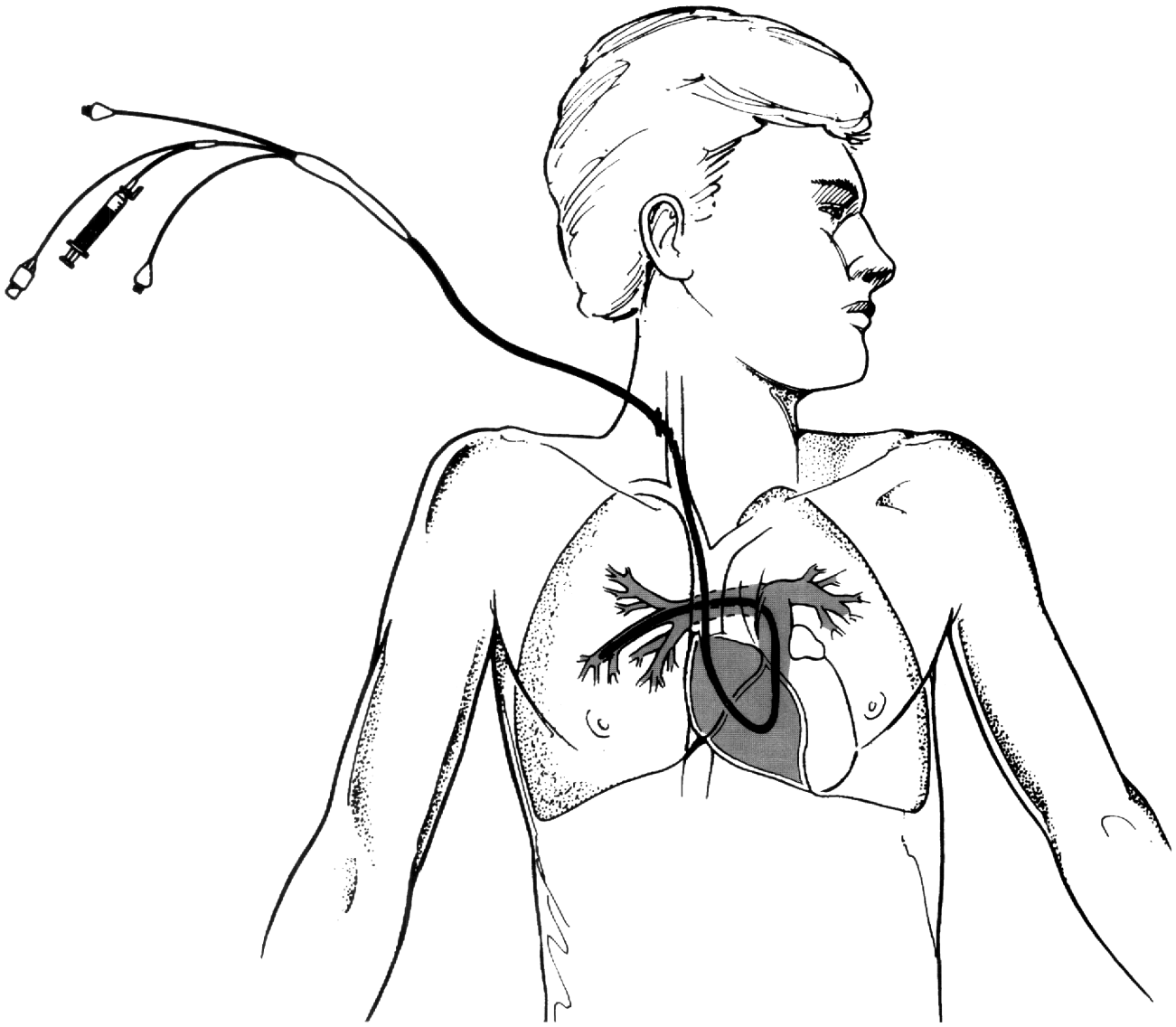
catheter reaches the pulmonary artery, the balloon can be inflated to occlude the artery and allow the downstream pulmonary venous pressure (wedge or occlusion pressure) to be measured from the distal lumen of the catheter. The proximal lumen of the catheter, located 30 cm from the tip, is positioned near the right atrium and is available for measurement of central venous pressure (CVP) and for injection of indicator solutions. A wire runs the length of the catheter, terminating in a thermistor 3.5 to 4.0 cm from the tip. This thermistor is used to determine cardiac output by thermodilution, a method that measures the temperature change caused by the injection of a cold indicator solution into the right atrium.

The introduction of the Swan-Ganz pulmonary artery catheter allowed cardiac output, for the first time, to be determined routinely at the patient's bedside, in the intensive care unit, or in the operating room.

Table 1.1 — History of Cardiac Catheterization and Measurement of Cardiac Output:[14] Significant Events

Year	Event
1844	Bernard: First catheterization of right and left ventricle of the horse.
1929	Forssman: First catheterization of human right ventricle.
1930	Kline: Catheterization of the right ventricle in 11 patients and cardiac output measured by Fick method.
1941	Cournard and Richards: Right heart physiology. Nobel Prize work.
1947	Dexter: Congenital heart work. First use of pulmonary artery wedge position.
1950	Zimmerman and Lason: Retrograde left heart catheterization.
1953	Seldinger: Percutaneous technique for right and left heart catheterization.
1959	Ross: Transeptal left heart catheterization.
1959	Sones: Selected coronary arteriography.
1970	Swan and Ganz: Introduction of balloon-tipped, flow directed catheter for thermodilution cardiac output and pulmonary wedge pressure.
1977	Gruentzig: Transluminal coronary angioplasty.

Figure 2.1 — A pulmonary artery catheter is shown along with the anatomical relationships to the heart, great vessels, and lungs.



2.0 THERMODILUTION TECHNIQUE FOR CARDIAC OUTPUT DETERMINATION

Cardiac output has been measured using a variety of techniques in which an indicator is injected into the circulation, and the subsequent dilution of the indicator is the basis for the determination of cardiac output. The dye dilution technique, described in Section 1.4, served as the only practical clinical measurement until the introduction of the Swan-Ganz pulmonary artery catheter made the thermodilution method widely available. The indicator for the thermodilution technique is a cold fluid (colder than the patient's blood), usually saline solution or 5% dextrose in water (D5W), either iced (0 to 5° C) or room temperature. The cold liquid is injected via the pulmonary artery catheter into the right atrium, where it mixes with venous blood and causes the blood to cool slightly (Figure 2.1). The cooled blood is ejected by the right ventricle into the pulmonary artery, where it passes a thermistor near the tip of the pulmonary artery catheter. The thermistor measures the change in blood temperature as the cooled blood travels past on the way to the lungs. The extent of cooling is inversely proportional to cardiac output.

2.1 *Calorie Deficit of the Indicator Solution*

Ten milliliters of saline at 15° C has approximately 200 fewer calories than at 35° C (blood temperature is assumed to be 35° C for the purpose of this example), one calorie representing the amount of heat required to raise the temperature of one ml of water by one °C. These 200 calories equal the calorie deficit of the indicator relative to the blood. Mixing cool saline with blood, of course, cools the blood. Because conservation of heat requires that the total heat must remain unchanged when two liquids mix, the temperature resulting from a mixture of saline (10 ml at 15° C) and blood (500 ml at 35° C) can be estimated as follows:

(°C blood x ml blood) + (°C saline x ml saline)
= °C mixture x ml mixture

or (35° C x 500 ml) + (15° C x 10 ml) = °C mixture x 510 ml

Solving for °C mixture:

Figure 2.2 — An analogy to thermodilution cardiac output determination: Blood at 35° C is flowing through a pipe. The blood first passes by a site for injection of thermal indicator, then past a thermistor.

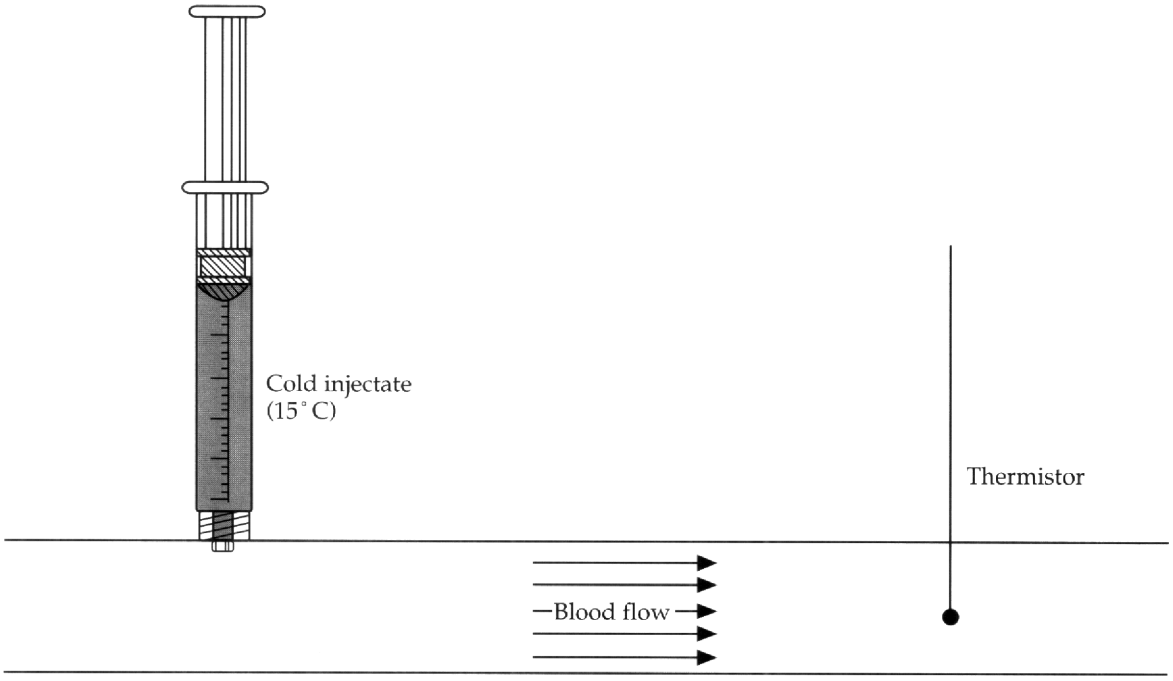


Figure 2.3 — An example of the thermodilution method for determination of cardiac output: Saline, 10 ml at 15° C, is injected over 5 seconds into blood flowing at 6 liters/minute. The saline mixes with 500 ml of blood (6000 ml/min x 1/12 min = 500 ml), which is cooled from 35° to 34.6° C.

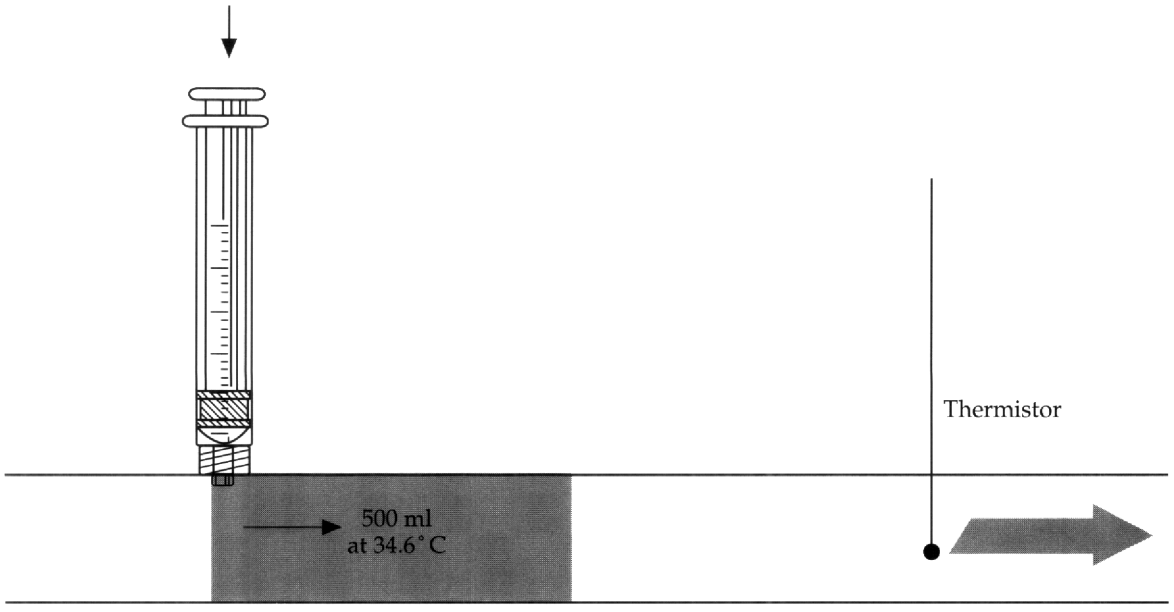
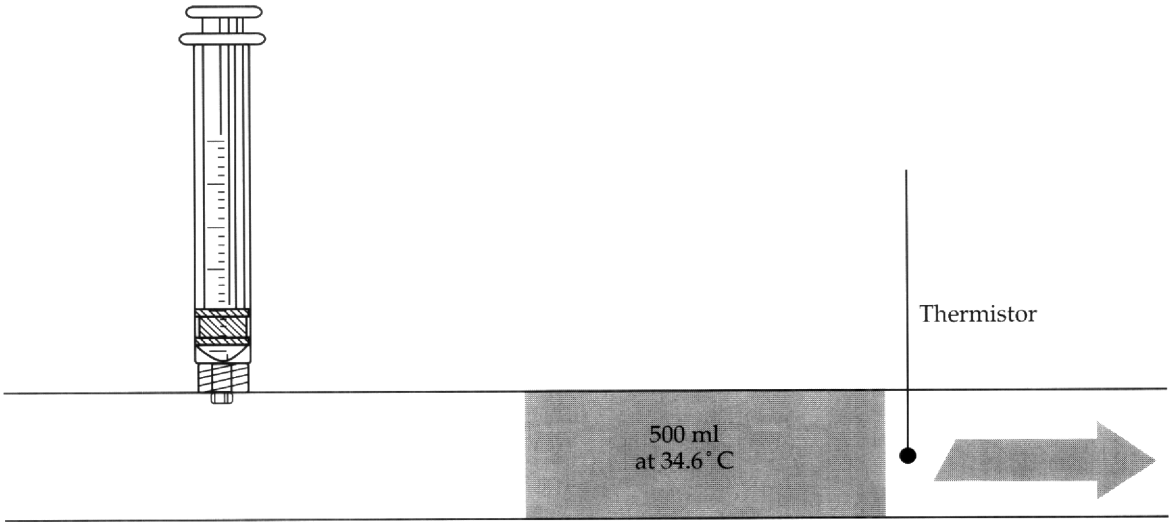


Figure 2.4 — With the thermodilution method of cardiac output determination, the cooled blood continues down the pipe to the thermistor, which registers a fall in temperature from 35° to 34.6° C, during the 5 seconds required for cooled blood to pass by the thermistor.



°C mixture ≅ 34.6

This calculation is actually not totally correct, because blood and saline have similar but not identical heat capacities and densities. When two liquids of different temperatures mix, the temperature of the mixture depends on the volume, the heat capacity, the density, and the temperature of each liquid. The factors for heat capacity and density are introduced into the calculation of cardiac output in Section 2.3.

2.2 Analogy to Thermodilution Cardiac Output

An analogy for the principles of the thermodilution technique is a pipe with blood running through it (Figure 2.2). The blood has a temperature of 35° C and flows at a rate of 6 liters/minute (6000 ml/min). Saline (10 ml at 15° C) is injected into the blood at a constant rate during a time interval of 5 seconds (Figure 2.3). The saline mixes with 500 ml of blood (1/12 min x 6000 ml/min = 500 ml) and this portion of blood cools to approximately 34.6° C.

When the blood (500 ml at 34.6° C) reaches the thermistor, the thermistor temperature falls 0.4° C and then returns to 35° C, 5 seconds later (Figure 2.4). The change in temperature may be plotted

Figure 2.5 — In the thermodilution method of cardiac output determination, thermistor temperature is plotted versus time. A decrease in temperature is recorded as a positive deflection, by convention. The shaded portion of the graph represents the area under the temperature-versus-time curve, a rectangle 0.4° C by 5 seconds (2 degree-seconds).

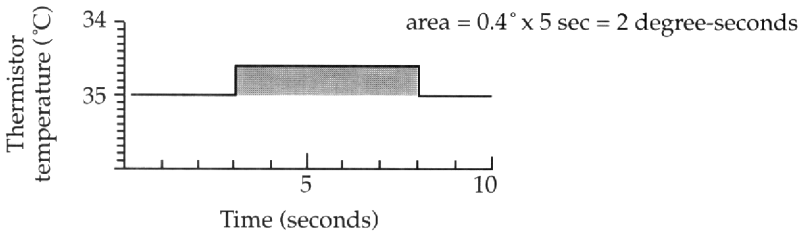


Figure 2.6 — Reducing the blood flow through the pipe by half causes the area under the curve to double: Saline, 10 ml at 15° C, is injected over 5 seconds into blood flowing at 3 l/min (instead of 6 l/min). Now the saline mixes with only 250 ml of blood and cools it to 34.2° C. When the cooled blood passes the thermistor, the 0.8° C drop in temperature is registered for 5 seconds to yield an area under the temperature-versus-time curve of 4 degree-seconds (instead of 2 degree-seconds when flow was 6 l/min).

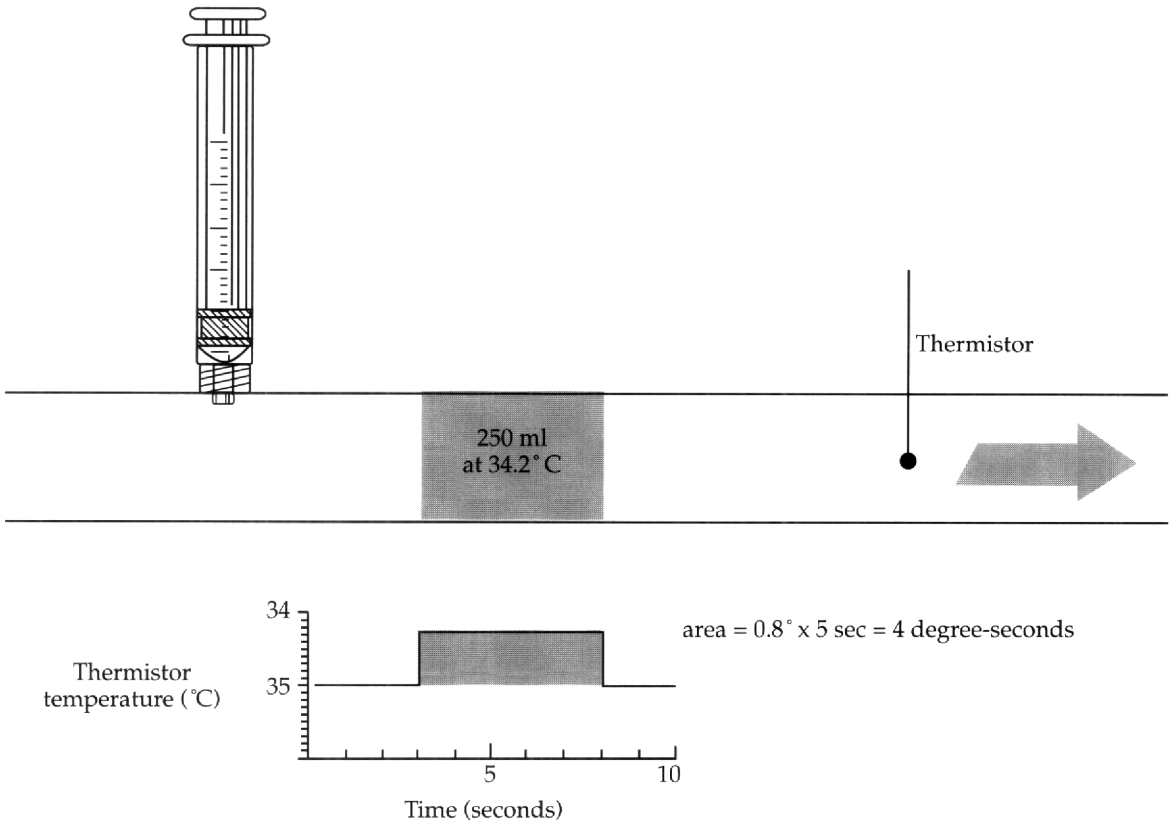
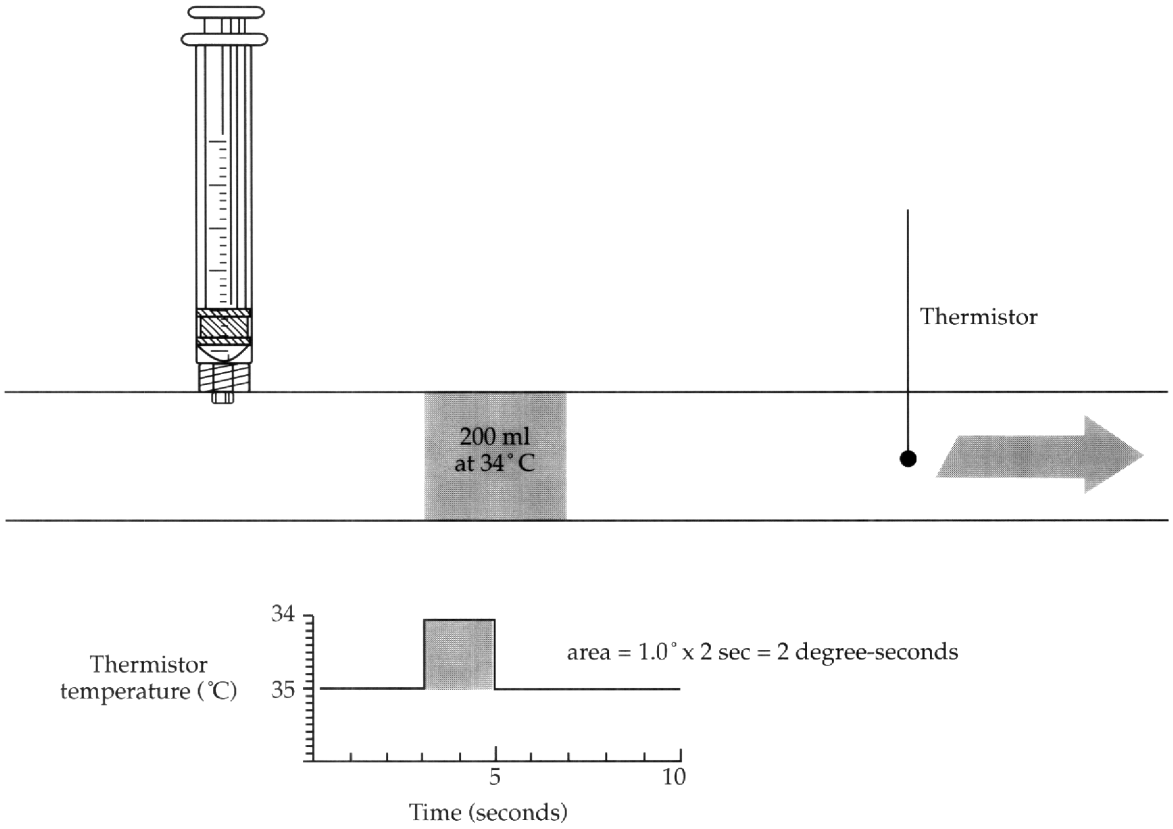


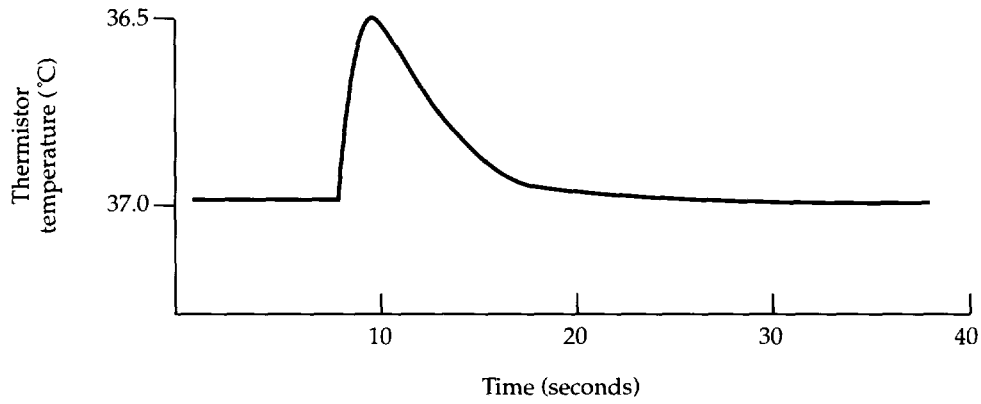
Figure 2.7 — The speed of injection of the cold indicator does not affect the area under the temperature-versus-time curve. In this example, saline, 10 ml at 15° C, is injected into blood flowing at 6 l/min, as in Figure 2.5. However, the injection is completed in 2 seconds instead of 5 seconds. The saline mixes with only 200 ml of blood (6000 ml/min x 1/30 min = 200 ml) instead of 500 ml, cooling the blood to 34° C instead of 34.6° C. When the cooled blood passes the thermistor, a temperature drop of 1° C is recorded for 2 seconds. The area under the temperature-versus-time curve is 2 degree-seconds, the same as in Figure 2.5.



with respect to time (Figure 2.5). By convention, a decrease in temperature is plotted as a positive deflection. The area under the temperature-versus-time plot is the product of the change in temperature multiplied by the time interval (in this case, 0.4° C x 5 seconds = 2 degree-seconds). The area under the temperature-versus-time plot serves as a useful parameter because it is inversely proportional to the rate of blood flow.

When the same saline indicator is injected into blood flowing at 3 l/min instead of 6 l/min, the saline mixes with 250 ml of blood (1/12 min x 3000 ml/min) instead of 500 ml of blood (Figure 2.6). The temperature drop is now 0.8° C instead of 0.4° C, and the area

Figure 2.8 — A typical thermodilution curve: A drop in thermistor temperature is recorded as a positive deflection, by convention. The upstroke is rapid and the decline to baseline blood temperature is monoexponential. The area under the temperature-versus-time curve is inversely proportional to the blood flow or cardiac output.



under the temperature-versus-time plot is 4 degree-seconds, twice as large as the previous example.

The area under the temperature-versus-time plot is not affected by the speed of injection of the cold liquid. When the saline is injected into blood flowing at 6 l/min during a 2-second period instead of a 5-second interval, the saline mixes with 200 ml of blood (1/30 min x 6000 ml/min) instead of 500 ml of blood (Figure 2.7). The blood is then cooled to 34° C instead of 34.6° C. The thermistor registers the 1° C temperature drop for 2 seconds. The area under the curve remains 2 degree-seconds (1° C x 2 seconds), the same as with a 5 second injection period. The product of blood flow and the area under the temperature-versus-time plot is equal to the calorie deficit of the injected indicator fluid:

$$\text{Calorie Deficit} \cong CO \times \int \Delta T \times dt$$

where CO = blood flow (cardiac output)
$\int \Delta T \times dt$ = area under the temperature-versus time plot.

In the examples above, the product of blood flow rate and area under the temperature-versus-time plot was 200° C–ml, the calorie deficit of saline (10 ml at 15° C) relative to blood at 35° C. For example, 6 l/min x 1 min/60 seconds x 1000 ml/l x 2 degree-seconds

= 200° C–ml or 200 cal, because cal = °C–ml (assumes that the density and specific heat of saline and blood are identical whereas, in fact, they are slightly different). The blood flow is proportional to the calorie deficit of the indicator fluid divided by the area under the temperature-versus-time plot:

$$CO \cong \frac{\text{Calorie deficit}}{\int \Delta T \times dt}$$

where CO = cardiac output
Calorie deficit = difference in heat content between the indicator fluid and the blood
$\int \Delta T \times dt$ = area under the temperature-versus-time plot.

Thus, if temperature and volume of the injected indicator are known and the area under the temperature-versus-time plot is measured, the blood flow can be calculated.

2.3 Actual Thermodilution Temperature-Versus-Time Curves

Real thermodilution temperature-versus-time plots are curves, not square waves as shown in the previous analogy. This is primarily because the right ventricle does not completely empty with each contraction. Some cooled blood remains in the ventricle at the end of systole; this residual mixes with more blood as the ventricle refills. With each successive contraction, some of the remaining cold blood leaves the right ventricle and passes by the pulmonary artery catheter thermistor. Furthermore, most commercial thermistors do not respond instantaneously to a new temperature, but have a time constant of 250 to 500 msec, resulting in a smoothing of the temperature-versus-time curve (Figure 2.8).

Even though the temperature curve no longer forms a square wave, the principle of conservation of heat still applies—i.e., the heat lost by the blood must equal the heat gained by the cold injectate (calorie deficit):

$$V_{inj} \times (T_{inj}-T_{blood}) \times C_{inj} \times D_{inj} = CO \times C_{blood} \times D_{blood} \times \int \Delta T \times dt$$

where V_{inj} = volume of injectate
T_{inj} = temperature of injectate
T_{blood} = baseline blood temperature
C_{inj}, C_{blood} = specific heat of injectate or blood (calories/g/°C)
D_{inj}, D_{blood} = density of injectate or blood (g/ml)
CO = cardiac output (ml/sec)
$\int \Delta T \times dt$ = area under temperature-versus-time curve (°C x seconds).

Solving the equation for cardiac output yields the Stewart-Hamilton equation:[15, 16]

$$CO = \frac{V_{inj} \times (T_{inj} - T_{blood}) \times C_{inj} \times D_{inj}}{C_{blood} \times D_{blood} \times \int \Delta T \times dt}$$

2.4 Correction for Injectate Warming

The Stewart-Hamilton equation assumes that no heat exchange occurs between the injected indicator solution and the pulmonary artery catheter. However, this is not a valid assumption because the cold indicator tends to gain heat as it passes through the pulmonary artery catheter, which is relatively warm.

Between the site of injection of the indicator and the point where the catheter enters the patient, the temperature of the catheter reflects ambient room temperature. The portion of catheter within the patient's body reflects the patient's core temperature. When a cold indicator is injected into a pulmonary artery catheter that is in thermal equilibrium with its surroundings, the first fluid to enter the right atrium is the warm fluid already in the catheter (the residual volume is <1 ml). The cold injectate then enters the right atrium, having warmed slightly during transit through the pulmonary artery catheter. The volume of cold injectate reaching the right atrium is reduced by the residual volume of the catheter. At the end of the injection, the plastic material of the pulmonary artery catheter has cooled and the catheter lumen is full of cold injectate.

Thus, the actual amount of thermal indicator that reaches the right atrium is reduced by the warming which takes place in the pulmonary artery catheter and by the residual volume of the catheter. The first bolus of a rapid series of injections is most affected by this phe-

nomenon because the fluid in the catheter is nearest to thermal equilibrium with the patient. Subsequent closely spaced injections are less affected because the pulmonary artery catheter and its residual fluid are still cold from the previous boluses of cold indicator.

A variety of tactics have been used to minimize the thermal effects of the catheter on the injected indicator. One method employs aspiration of the fluid in the catheter immediately before and after an injection.[17] This technique provides for the most consistent transfer of cold, but it is quite cumbersome. Another procedure discards the first of a series of injections, because the greatest loss of thermal indicator occurs during the first bolus, which would result in an overestimation of true cardiac output.

Manufacturers of pulmonary artery catheters have empirically determined the extent of heat transfer from the catheter to the cold indicator under specified conditions.[13] A correction factor (F) that is based upon such data was incorporated into the version of the Stewart-Hamilton equation introduced by Ganz and Swan,[18] and appears in the software of all commercially available thermodilution cardiac output computers:

$$CO = \frac{V_{inj} \times (T_{inj} - T_{blood}) \times C_{inj} \times D_{inj} \times F}{C_{blood} \times D_{blood} \times \int \Delta T \times dt}$$

where F = correction factor.

The factor F can be considered the fraction of the calorie deficit in the injectate that reaches the thermistor during a cardiac output determination and is always less than 1.

The correction factor used to account for the warming of the injectate must be measured separately for each brand of catheter because of differences in catheter tubing material, lumen size, and wall thickness. Correction factors are empirical and are determined under specific conditions. Typical examples of these conditions include:

- 25 to 45 cm portion of catheter inside the patient.
- Injections made every 1 to 2 minutes.
- The cardiac output determined from the initial injection of a series is discarded.
- 2.5 to 5.0 ml/sec rate of injection (10 ml in 2 to 4 seconds).

- A narrow range of injectate temperature, either iced (0 to 4° C) or room temperature.
- Specified injectate volume of either 3 ml, 5 ml, or 10 ml.

2.5 Calculation of Cardiac Output by Computer

Commercially available thermodilution cardiac output computers use the Stewart-Hamilton equation. The six constants V_{inj}, C_{inj}, D_{inj}, F, C_{blood}, and D_{blood} are combined with the proportionality constants 60 sec/min and 1 liter = 1000 ml into a single constant (with units of 1 liter-sec/min), which is commonly referred to as the computation constant:

$$CO = \frac{V_{inj} \times C_{inj} \times D_{inj} \times F \times 60 \text{ sec/min} \times 1 \text{ l}/1000 \text{ ml}}{C_{blood} \times D_{blood}} \times \frac{(T_{inj} - T_{blood})}{\int \Delta T \times dt}$$

where

CO	=	cardiac output (l/min)
V_{inj}	=	3 ml, 5 ml, or 10 ml
C_{inj}	=	0.965 calories/g/°C (D5W; saline is 0.997, a small difference which is ignored)
D_{inj}	=	1.018 g/ml (D5W; saline is 1.005, a small difference which is ignored)
F	=	depends upon specific conditions; ranges from 0.865 to 0.938 for 10 ml injectate volume
C_{blood}	=	0.87 calories/g/°C
D_{blood}	=	1.045 g/ml.

The computation constant appears in the specific pulmonary artery catheter package insert. Different constants exist for iced and room temperature injectate (F varies) and for 3 ml, 5 ml, and 10 ml injectate volumes (V_{inj} varies). The appropriate constant is entered into the cardiac output computer by the operator.

The variables in the Stewart-Hamilton equation that must be measured, therefore, are T_{inj}, T_{blood} and $\int \Delta T \times dt$:

$$CO = \text{Combined Constant} \times \frac{(T_{inj} - T_{blood})}{\int \Delta T \times dt}$$

When the operator initiates a cardiac output determination, the computer begins to monitor the temperatures of the pulmonary artery and injectate thermistors. Injectate temperature is measured at a single point in time, usually by a specific injectate thermistor located directly in the injectate fluid pathway (see Section 3.2). The baseline blood temperature is recorded by the pulmonary artery catheter thermistor. Baseline blood temperature is averaged over a 2-second period to smooth out fluctuations that commonly occur due to ventilatory variation in pulmonary artery blood temperature.

The area under the temperature-versus-time curve ($\int \Delta T \times dt$) is determined by electronic integration. The downsloping portion, or tail, of the curve declines exponentially[6, 10] and must be integrated to an infinite time (∞). Because temperature is not actually measured for an infinite time, the area under the tail of the curve must be calculated based on the exponential nature of the decline in temperature. Several different algorithms exist for this purpose. One method determines the time constant (K) for the exponential decline (Figure 2.9). The area under the tail is then calculated by:

$$AUC_{tail} = T \times K$$

where AUC_{tail} = area under the temperature-versus-time curve for the tail portion of the curve

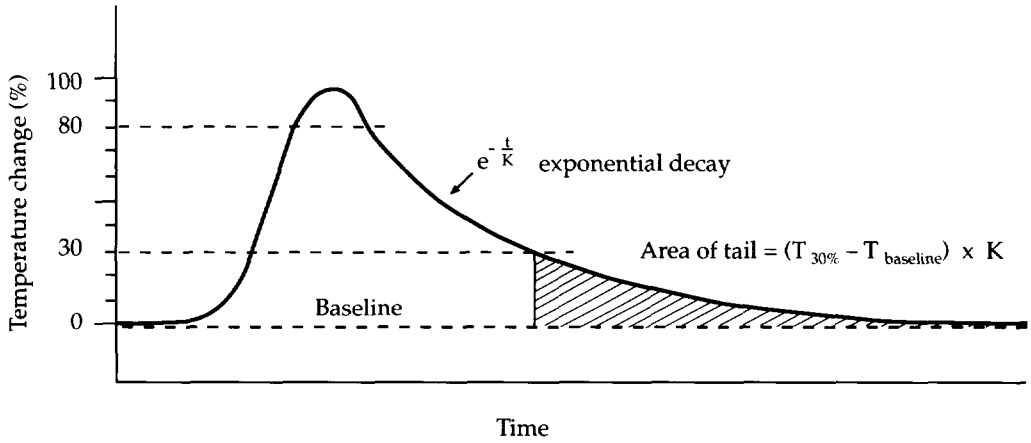
T = temperature deflection at the designated starting point of the tail portion of the curve

K = time constant for monoexponential decline of temperature.

The area under the tail of the curve is added to the area under the curve which precedes the tail portion to give the total area under the curve. A common algorithm begins calculating the tail at the point where the temperature deflection declines to 30% of peak value (Figure 2.9).

Another method used to determine area under the tail portion of the temperature-versus-time curve considers the empirical fact that, on the tail portion of the curve, the area between 75% and 37.5% of the maximum deflection is equal to the area from 37.5% of the maximum deflection to infinity. Integration of the area under the

Figure 2.9 — An algorithm for calculating the area under the tail of the temperature-versus-time curve. The area of the tail of the curve (shaded) must be calculated from the time constant (K) for the monoexponential decline of temperature. The computer determines the time constant between 80% and 30% of the peak temperature deflection. The area of the tail is the time constant, K, times 30% of the peak temperature deflection.



curve between 75% and 37.5% of maximum deflection, multiplied by two, yields the area under the curve from 75% of the maximum deflection to infinity.

2.6 Continuous Thermodilution Cardiac Output

In principle, heat could be used as a thermodilution indicator instead of cold. Incorporation of a heating filament into a pulmonary artery catheter has been proposed as a method to introduce heat without the need to inject fluid. However, the filament surface temperature must be limited (a maximum temperature of 44° C has been recommended)[19] in order not to cause thermal damage to blood or tissues. Because the amount of heat that can be introduced safely is relatively small, the "signal to noise" ratio is quite low. The normal variations in pulmonary artery blood temperature that occur with the respiratory cycle contribute to the "noise" level (Section 3.1). A solution to the "signal to noise" problem has been attempted by the use of stochastic techniques in which heat is supplied to a catheter-mounted filament according to a pseudorandom binary sequence.[20] With this approach, Yelderman and co-workers developed the means to deter-

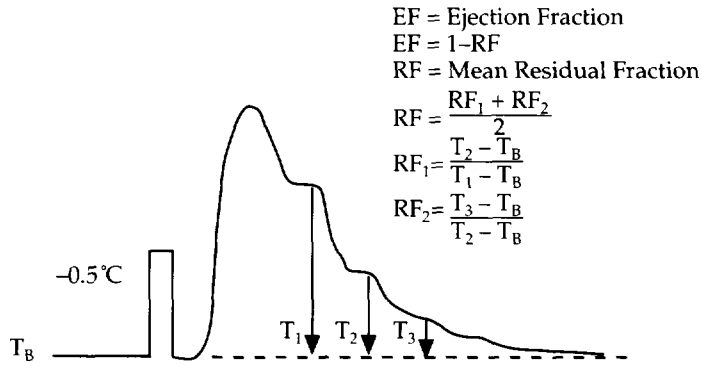
mine thermodilution cardiac output "continuously," using heat from a catheter-mounted filament as the indicator. The displayed cardiac output is updated every 30 seconds based on the average output of the previous 3 to 6 minutes. The correlation coefficient between this technique and conventional thermodilution cardiac output, determined from bolus injection of cold indicator fluid, was r = 0.94.[21]

2.7 Right Ventricular Ejection Fraction by Thermodilution

The possibility of determining ventricular volumes and ejection fraction by thermodilution has been recognized for many years.[22] When a rapid-response thermistor is employed to measure pulmonary artery temperature, the downslope of the washout curve appears as a series of small steps rather than a smooth curve, each step corresponding to an individual heart beat. Each successive change in temperature represents ejection of progressively warmer blood from the right ventricle as cold indicator is diluted with warmer blood flowing in from the right atrium. The ratio between 2 successive steps estimates the fraction of ventricular end-diastolic volume that remains after systole, the "residual fraction" (RF) (see Figure 2.10). Ejection fraction (EF) is calculated as: EF = 1 - RF. Right ventricular stroke volume (SV) is calculated by dividing the cardiac output (determined by thermodilution) by heart rate: SV = CO/HR. Right ventricular end-diastolic volume (EDV) is then calculated by dividing stroke volume by ejection fraction: EDV = SV/EF. Right ventricular end-systolic volume (ESV) is calculated by subtracting stroke volume from end diastolic volume: ESV = EDV - SV.[22]

There are certain limitations to the accuracy of this technique. Intracardiac shunting, tricuspid regurgitation, or irregular heart rhythms can produce errors. Embedding a rapid-response thermistor in a pulmonary artery catheter slows the response time. The thermistor must respond completely to a change of temperature within the interval between heart beats. The error caused by failure of the thermistor to equilibrate, underestimation of ejection fraction, is magnified at higher heart rates. Thus, computer algorithms for calculating ejection fraction and ventricular end-diastolic volume must compensate for the effect of heart rate.[23]

Figure 2.10 — Method for calculating ejection fraction by thermodilution. A fast response thermistor is used. The plateaus on the downsloping portion of the curve represent cardiac diastole. T_1, T_2, and T_3 are the differences between baseline temperature (T_B) and the temperature during diastole of three successive heart beats.



The accuracy of right ventricular ejection fraction determined by thermodilution has been tested by comparison to other methods, such as ventricular angiography and radionuclide imaging techniques. The reported correlation coefficients, while statistically significant, do not generally approach unity (r ranged from 0.45 to 0.85).[23]

The clinical utility of measuring right ventricular ejection fraction at the bedside is unclear. Treatment of clinical conditions that place a stress on the right ventricle, such as pulmonary hypertension, adult respiratory distress syndrome (ARDS), and myocardial infarction involving the right ventricle, might be enhanced by measuring right ventricular function by thermodilution.

3.0 ACCURACY OF THERMODILUTION CARDIAC OUTPUT: COMMON PROBLEMS AND SOURCES OF ERROR

The overall accuracy and reproducibility of thermodilution cardiac output has been verified repeatedly by comparison to other techniques, including the Fick method, dye dilution, and electromagnetic flow measurement (Figure 3.1). Variation of serial thermodilution cardiac output measurements is generally 5% or less under ideal circumstances and correlation to other standard techniques results in

correlation coefficients of greater than 0.9.[24] Since the error of a single determination of cardiac output may be somewhat larger than 5%, the usual practice is to average the results of three cardiac output determinations (typically 10 ml of room temperature solution injected at a constant rate during a 2-4 second period). If injectate volumes of less than 10 ml are used, the solution should be iced. To obtain optimal results one must be aware of pitfalls that may result in relatively larger errors.

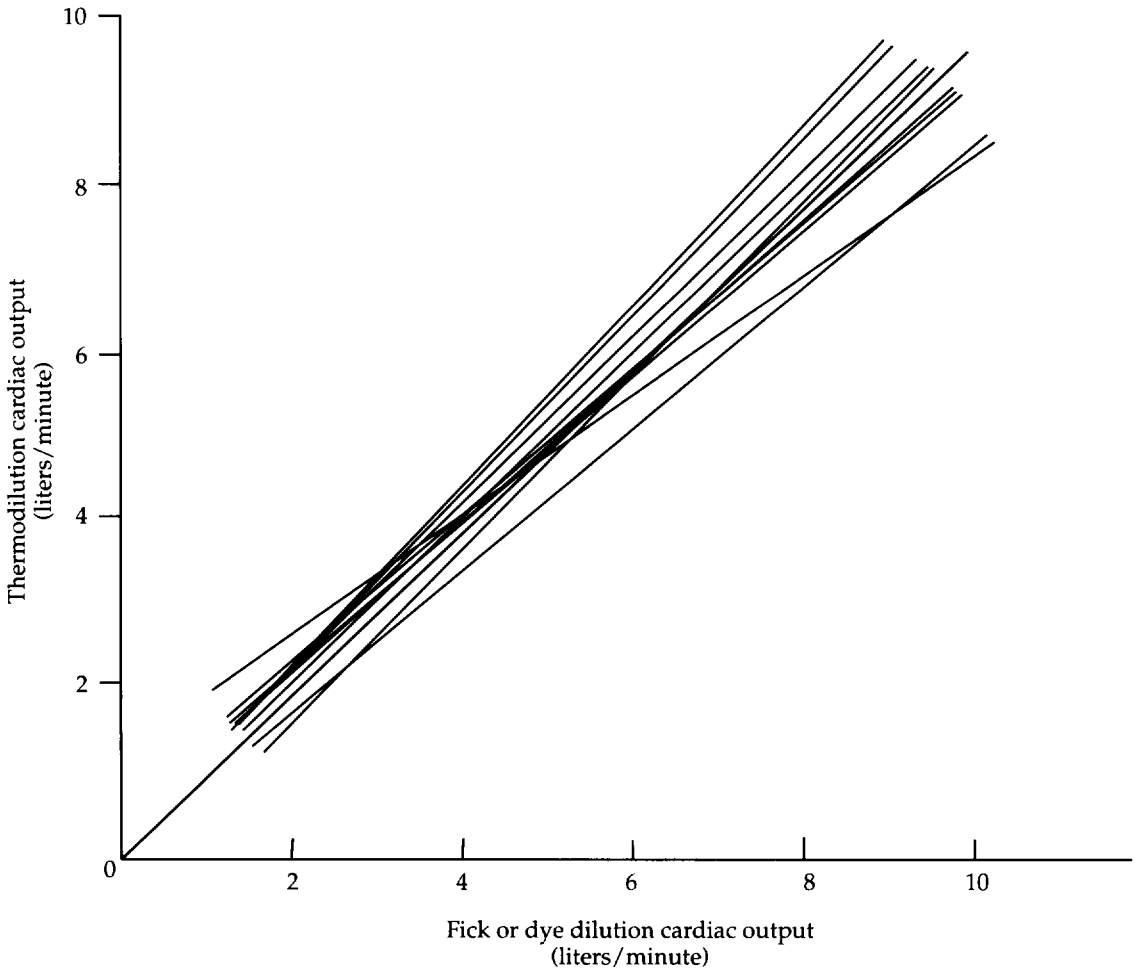
3.1 *Timing the Thermodilution Injection During the Ventilatory Cycle*

Thermodilution cardiac output measurement relates to the ventilatory cycle in at least two distinct ways. First, baseline pulmonary artery temperature undergoes cyclic variation with the ventilatory cycle. Second, the stroke volume (CO = SV x HR) varies during the ventilatory cycle, probably because of changes in venous return and right ventricular afterload.

The calculation of thermodilution cardiac output depends on the difference between baseline pulmonary artery blood temperature and injectate temperature (T_{inj} - T_{blood}). Studies in animals and humans have demonstrated phasic variation in pulmonary artery temperature that relates to the ventilatory cycle.[25] The temperature of venous blood varies in different regions of the body. The proportion of blood from each region entering the vena cava fluctuates with the ventilatory cycle, resulting in cyclic temperature variation in the right atrium and pulmonary artery (Figure 3.2). The magnitude of these temperature changes occurs in the range of 0.01 to 0.10° C, resulting in artifact (noise) in the determination of baseline blood temperature (T_{blood}) and introducing the possibility of a small error in the measurement of cardiac output.

The signal-to-noise ratio can be improved by maximizing the difference in temperature between blood and injectate. Wessel has estimated that two- to three-fold improvement occurs in signal-to-noise ratio by the use of iced injectate (0 to 4° C) instead of room temperature injectate.[26] However, in clinical practice this is not a crucial consideration, provided the volume of room temperature injectate is not less than 10 ml. Elkayam and colleagues studied critically ill patients in the intensive care unit and found that 10 ml of room tem-

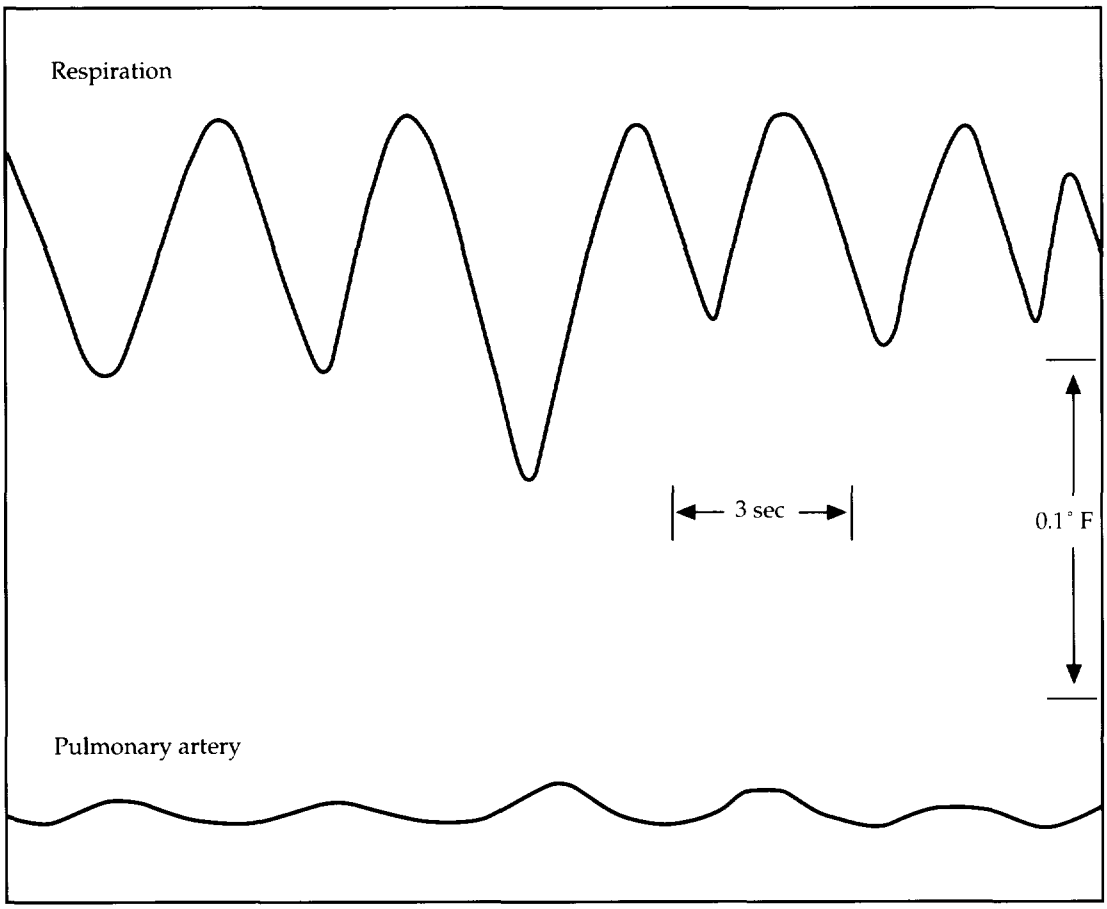
Figure 3.1 — The correlation between Fick or dye dilution cardiac output and thermodilution cardiac output has been examined repeatedly. This figure summarizes the results of 13 different studies. The regression line for the linear relationship between thermodilution and Fick or dye dilution cardiac output technique is plotted for each study. The dark line represents the average of the results of all of these studies and has a slope equal to 1.



perature injectate gave results comparable to 10 ml or 5 ml of iced injectate.[27] The use of 5 ml of room temperature injectate was associated with a substantial reduction in reproducibility of cardiac output measurements.

The effect of variation in baseline temperature for cardiac output determination can also be minimized by timing each injection to start at the same point in the ventilatory cycle. Several studies have

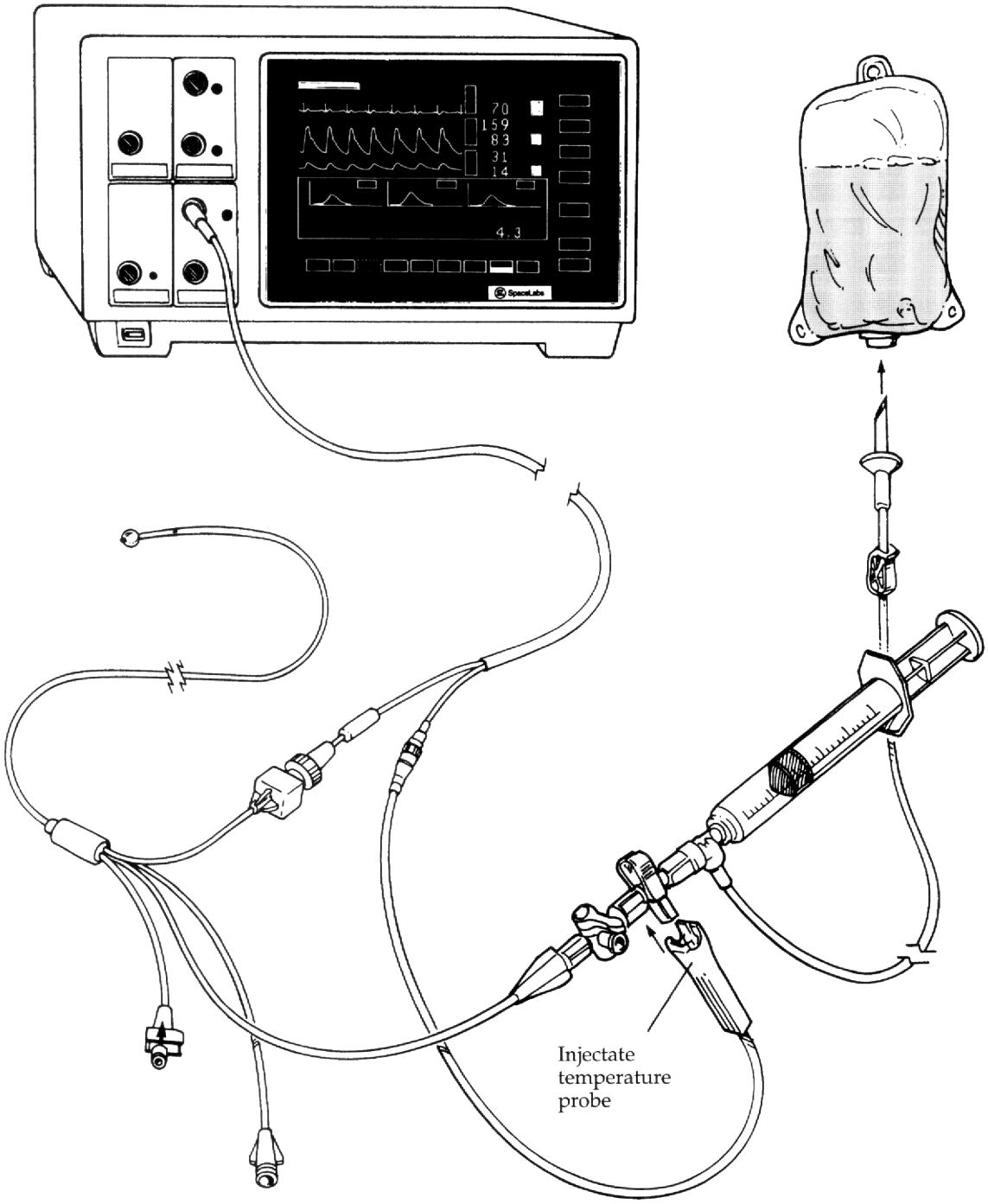
Figure 3.2 — Pulmonary artery temperature varies slightly with the respiratory cycle, as shown in this illustration of a patient breathing spontaneously. The upper tracing records respiration, while the lower tracing represents the simultaneous pulmonary artery temperature.



documented improved reproducibility of cardiac output determinations performed in this fashion.[28, 29]

Timing of thermodilution injection is further complicated by the fact that stroke volume may change with respiration. Snyder and Powner found that stroke volume and thermodilution cardiac output varied substantially during the ventilatory cycle in mechanically ventilated dogs, presumably because of changes in venous return and right ventricular afterload.[30] Therefore, timing each injection to the same point in the ventilatory cycle could be misleading because the true cardiac output is the average output

Figure 3.3 — The most accurate method for measuring injectate temperature is at the point of injection using the apparatus shown. The injectate fluid is drawn from a bag into a syringe through a length of tubing and a one-way valve. When the fluid is injected, it flows past a thermistor that is placed directly in the fluid pathway, between the syringe and the pulmonary artery catheter. The thermistor is connected to the cardiac output computer, which measures that temperature and enters the data into the algorithm for computing cardiac output.



during the entire ventilatory cycle. These authors recommend that cardiac output be determined by the mean of several values measured at "regularly spaced intervals through the ventilation cycle."

The timing of thermodilution injection relative to the ventilatory cycle remains a remarkably complex physiological problem. However, in clinical practice, detection of significant changes in cardiac output is more important than a few percentage points of error in the measurement itself. Performing the injection in the same way each time cardiac output is determined is most important in accurately tracking alterations in cardiac output. Reasonable options for consistent timing of injection include:

- Inject at the end of expiration or at some other constant point in the ventilatory cycle.
- Make several injections at random times with respect to the ventilatory cycle and average the results.

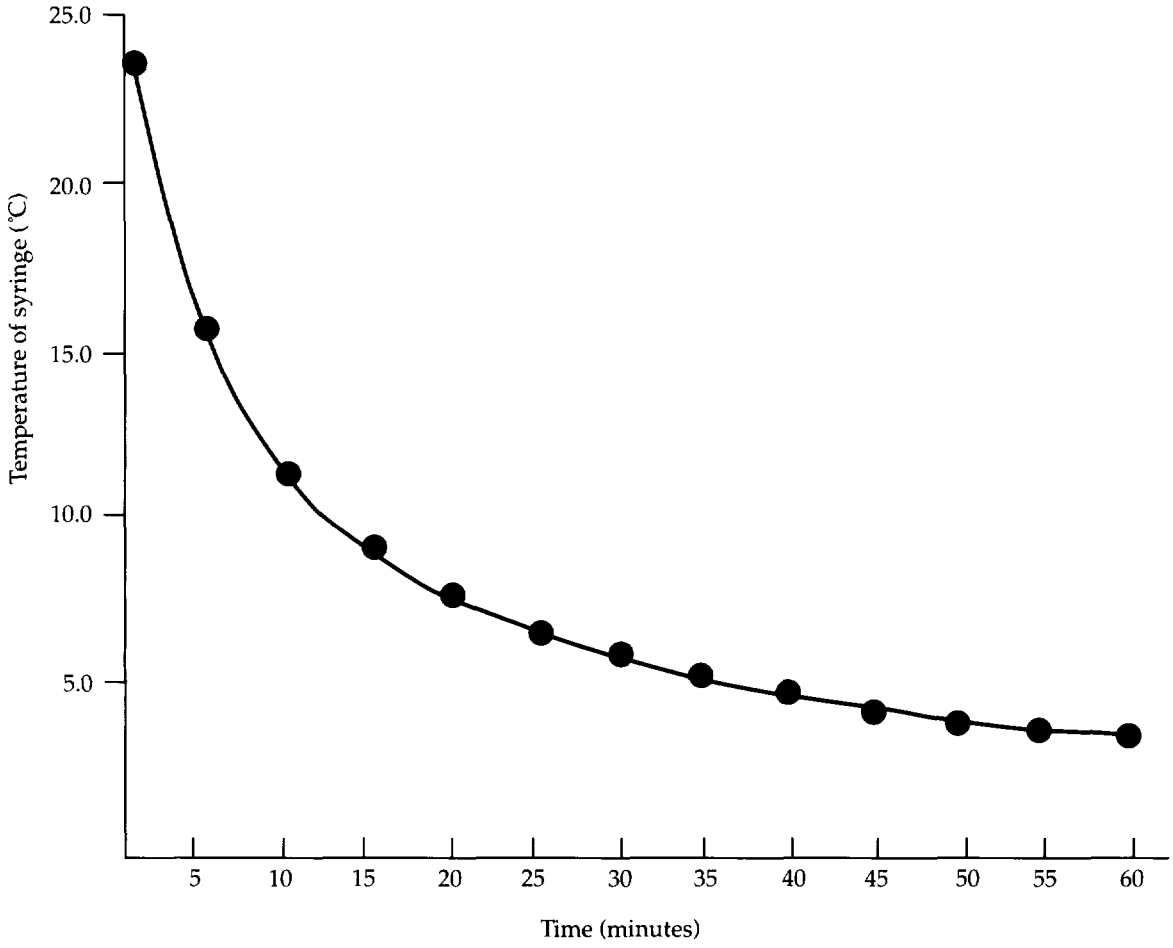
3.2 Measurement of Injectate Temperature

Current technology allows the accurate measurement of injectate temperature at the point of injection (Figure 3.3). In the past, a temperature probe, connected to the cardiac output computer, was placed into an iced bath containing the syringes of injectate. However, this practice resulted in several problems. The temperature in the ice bath varies with the level at which the probe is placed; thus the probe should be inserted at the same level as the syringes. Because the probe measures the temperature of the bath, and not of the syringes themselves, the syringes must remain in the bath long enough to equilibrate.[24] Plastic 10 ml syringes filled with 5 ml of D5W at room temperature require approximately 60 minutes to reach steady state in an ice bath at 0 to 30° C (Figure 3.4).[24] Cold syringes begin to warm immediately when removed from the ice bath and handled prior to injection. However, this rewarming is relatively insignificant (about 0.5° C) if injection occurs within 30 seconds.[31]

3.3 Pulmonary Artery Thermistor Position

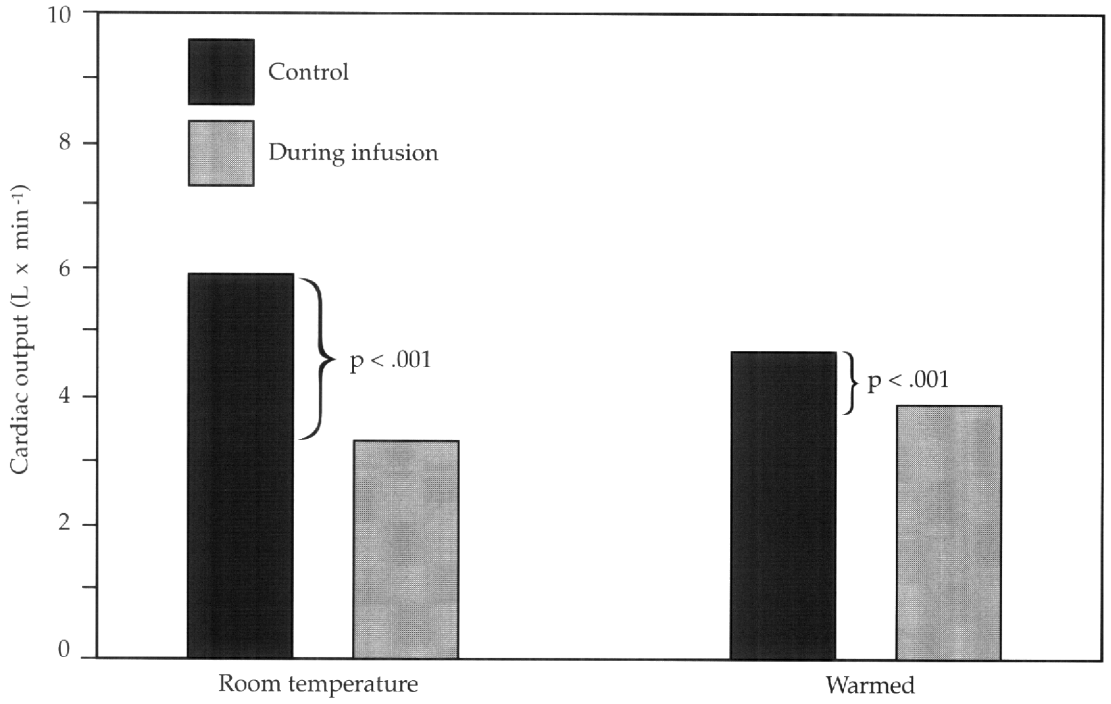
Thermal gradients can exist between blood flowing in the center of an artery compared to blood flowing near the vessel's edge, the latter being more affected by the thermal properties of the vessel

Figure 3.4 — In the past, syringes containing injectate for thermodilution cardiac output were cooled in an ice bath. The injectate temperature was measured indirectly by a thermistor placed in the ice bath. As shown in this illustration, an hour or more is required to reach temperature equilibrium between the syringes and the ice bath. If the syringes are used prior to equilibrium, a significant error can occur since the actual injectate temperature will be higher than the temperature measured in the ice bath.



wall.[32] The ideal position for the thermistor, to most accurately detect temperature changes caused by the injectate, is near the center of the vessel. In clinical practice, thermistor positions between the pulmonary valve and peripheral pulmonary artery branches produce comparable thermodilution cardiac output results, as long as the pulmonary artery pressure trace is not damped, which indicates that the catheter tip may be in contact with the vessel wall.[33, 34] The absence of damping is determined by visual inspection of the pulmonary artery pressure trace.[35]

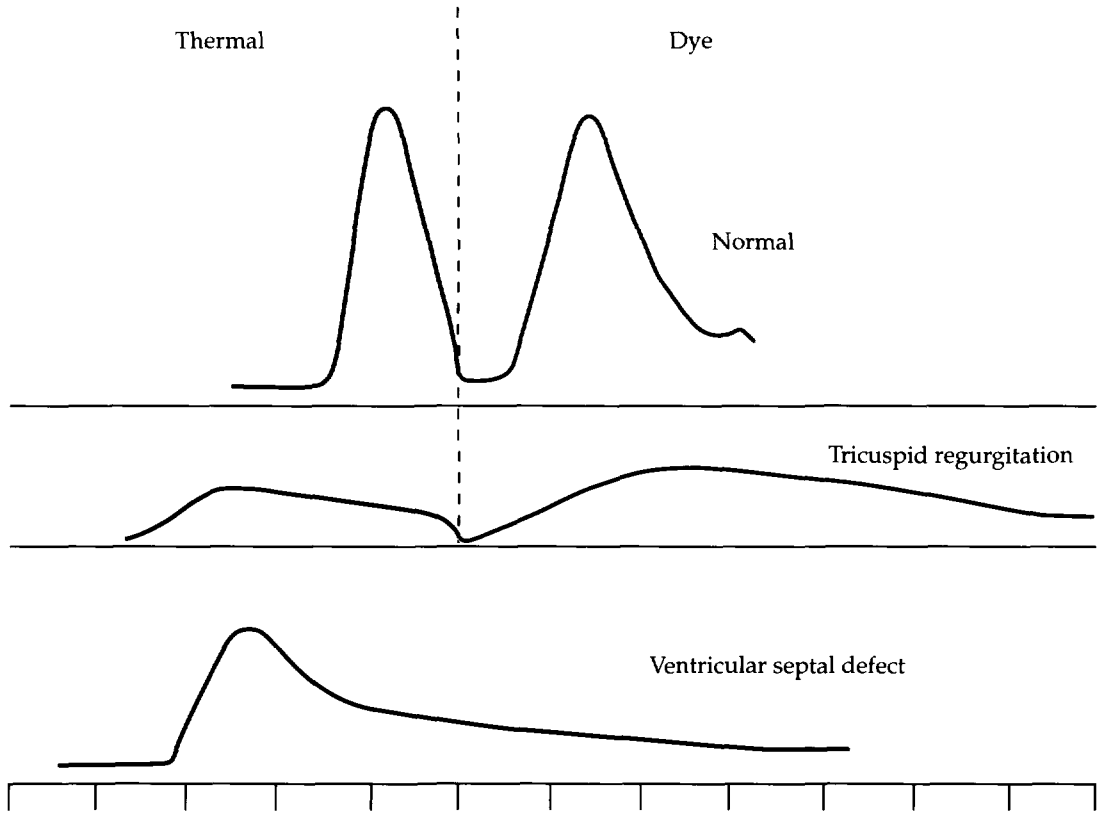
Figure 3.5 — Rapid intravenous infusion of fluids can cause errors in thermodilution cardiac output determination. Shown here is the effect of infusing room temperature or warmed fluids into a peripheral intravenous line just prior to injection of the thermodilution indicator fluid. The measured cardiac output is significantly less than cardiac output determined under control conditions. The intravenous fluid cools the pulmonary artery thermistor, just as the thermistor modulation injectate does, resulting in a larger area under the thermodilution temperature-versus-time curve, and a smaller calculated cardiac output.



3.4 *Speed of Injection*

Although speed of injection does not appear as a discrete variable in the Stewart-Hamilton equation, it may have some effect on the empirical correction factor F because it affects heat transfer between the injectate and the catheter. However, Pavek compared injection at a constant rate of 1 to 2 ml/sec to a bolus delivered as fast as possible (instantaneous) and found no significant difference between the methods.[33] Swan and Ganz found that F was independent of injection rate between 2 and 4 seconds total injection time.[18] Thus, within these practical limits, speed of injection is not critical in thermodilution cardiac output measurements. While the exact speed of

Figure 3.6 — Intracardiac shunts can result in errors in cardiac output determination. The thermodilution or dye dilution curves under these conditions have an abnormal appearance. Compare the normal thermodilution and dye curves (top) to the curves obtained from patients with tricuspid regurgitation (center) or ventricular septal defect (bottom). The curve from the patient with tricuspid regurgitation is broad because blood in the right ventricle is injected into the right atrium as well as the pulmonary artery, causing the thermal or dye indicator to remain in the atrium and ventricle longer than usual. The downslope of the thermodilution curve from the patient with a ventricular septal defect is delayed because of recirculation of thermal indicator through the septal defect.



injection appears relatively unimportant, care should be taken that the injection rate remains constant. Because computer algorithms rely on the exponential decline of the thermodilution curve, uneven injection may result in erroneous calculations (see Section 2.5). Carbon dioxide-powered injection devices or guns deliver injectate at constant flow rates but are not necessary in routine clinical practice.

3.5 *Artifacts Caused by Intravenous Fluid Administration*

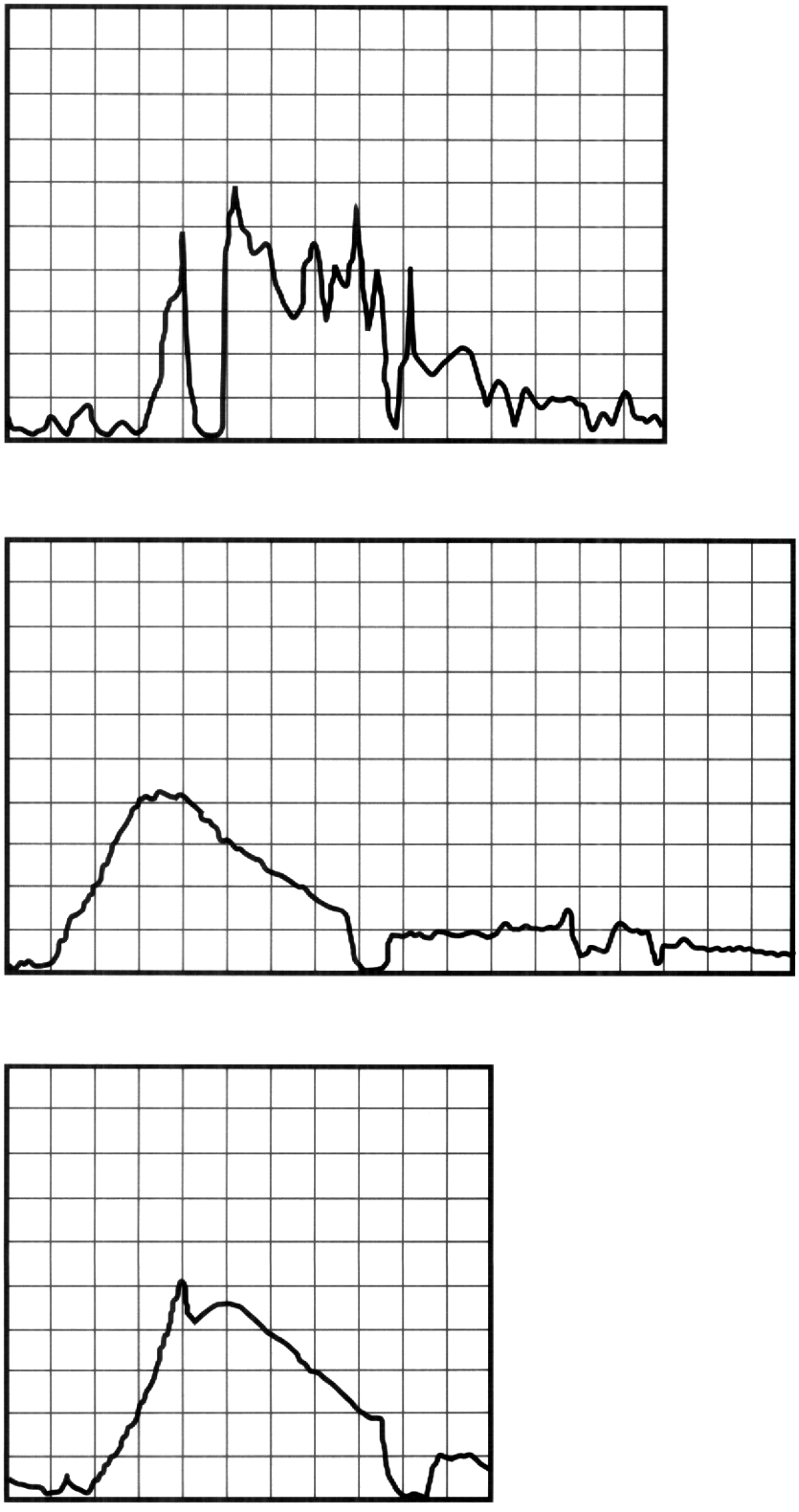
Wetzel and Latson demonstrated that bolus intravenous (IV) administration of room temperature or warmed fluids into an arm just prior to injection of thermodilution injectate caused a 20 to 80% reduction in the apparent cardiac output reading, from control values of cardiac output (Figure 3.5).[36] The magnitude of error was greatest with cold fluid boluses. Presumably, this error occurs because the rapid administration of intravenous fluid causes cooling of the thermistor, which combines with the cooling caused by the injectate and results in a larger area under the thermodilution temperature curve and a smaller calculated cardiac output. This type of error can happen easily in the critical care environment, especially the operating room, where intravenous fluids are commonly infused rapidly. Rapid administration of intravenous fluids might also cause errors because of instability of baseline pulmonary artery temperature.

3.6 *Bad Curves*

Visual inspection of the shape of the thermodilution curve should be routine because it can quickly reveal a variety of errors. The normal curve is smooth and is characterized by a rapid upstroke followed by a slower, exponential return to baseline. Cardiac output determinations associated with irregular curves or with unstable baselines should be discarded.

Two cardiac structural abnormalities that result in unreliable cardiac output determinations are tricuspid insufficiency and ventricular (or atrial) septal defects.[37] The thermodilution curve recorded from a patient with tricuspid insufficiency tends to be broad and flat because of the delay in washout of injectate from the site of injection in the right atrium into the pulmonary artery. A ventricular

Figure 3.7 — Cardiac output curves reflecting problems of electrosurgical units or of mechanical origin.



septal defect produces a prolongation of the downsloping portion of the curve due to recirculation of cooled blood through the septal defect (Figure 3.6).

An electrosurgical unit (ESU) emits radio frequency energy that can disrupt or scramble the thermodilution temperature curve and result in wildly inaccurate results (Figure 3.7). Such a disruption can cause major problems for the anesthesiologist measuring cardiac output intraoperatively. The effect of an ESU becomes readily apparent by inspection of the thermodilution temperature curve, another reason for the routine display of the curve. During cardiopulmonary bypass for cardiac surgery, marked hypothermia (20 to 32° C) is commonly induced to cool the heart and reduce the metabolic demand for oxygen. The patient is rewarmed prior to restarting the arrested heart and discontinuing bypass. Immediately following discontinuation of cardiopulmonary bypass, dramatic instability of the baseline pulmonary artery temperature may occur which effectively precludes the accurate determination of cardiac output. Presumably, this happens because of significant variation in the temperature of blood returning to the heart from regions of the body that have not fully rewarmed. The shape of the temperature curve therefore appears erratic.

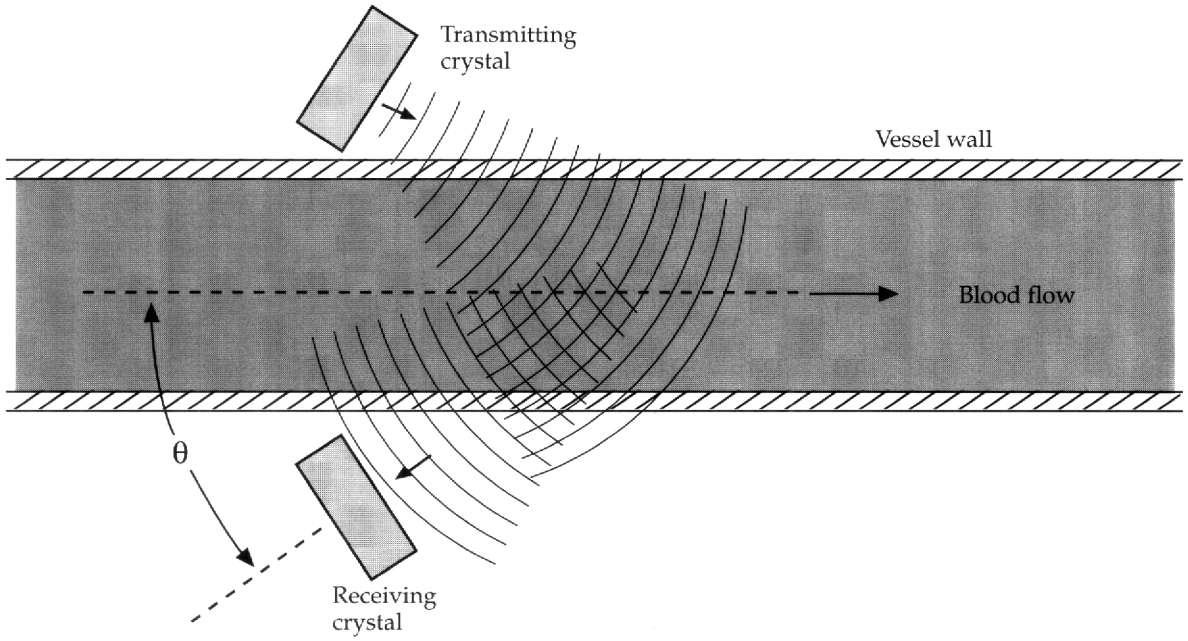
4.0 THE USE OF ULTRASOUND FOR CARDIAC OUTPUT MEASUREMENT

Ultrahigh frequency sound (ultrasound) can be used to measure the velocity of blood flow in the ascending aorta by application of the Doppler principle.[38] If the cross-sectional area of the aorta is known, or can be measured, the blood flow rate can be calculated simply as:

$$\begin{aligned} \text{Blood Flow} &= \text{velocity (cm/sec) x area (cm}^2\text{)} \\ &= \text{cm}^3\text{/sec} = \text{ml/sec x 1/1000 ml x 60 sec/min} \\ &= \text{l/min} \end{aligned}$$

The blood flow in the ascending aorta is identical to cardiac output minus the quantitatively negligible flow to the coronary arteries. The equation above is an oversimplification because blood flow in the aorta is not constant but pulsatile and blood velocity in the aorta

Figure 4.1 — The ultrasonic Doppler shift flow meter: Sound is generated by the transmitting crystal and reflected from moving red blood cells to the receiving crystal. The shift in frequency (Doppler shift) is directly proportional to the velocity of blood flow.



varies during the cardiac cycle. Ultrasound cardiac output devices actually measure the stroke volume by integrating blood velocity during cardiac systole:

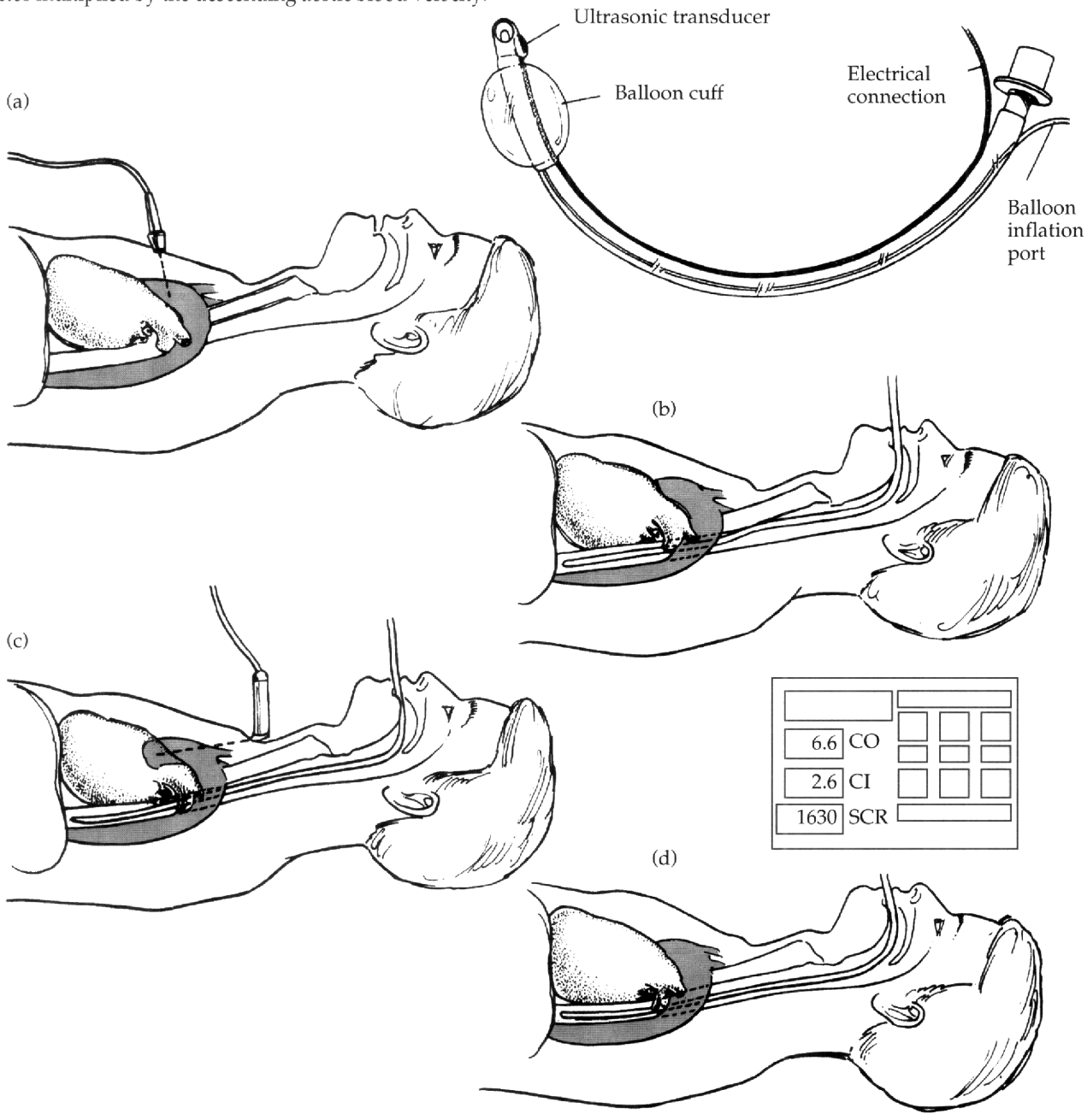
$$SV = CSA \times \int^{VET} V(t)\, dt$$

where
- SV = stroke volume
- CSA = cross-sectional area of the aorta
- VET = ventricular ejection time
- V = blood velocity.

Stroke volume is then multiplied by the heart rate to give cardiac output.

The Doppler principle describes a shift in sound frequency that occurs when sound emitted from a stationary transducer reflects off a moving object and returns to the transducer (Figure 4.1). The shift in frequency is directly proportional to the velocity of blood flow. The moving objects in the case of blood flow measurement are the red blood cells. Velocity is calculated from the Doppler equation:

Figure 4.2 — (a) Endotracheal tube with ultrasound probe at its tip for measurement of blood velocity in the pulmonary artery. Preoperative determination of aortic diameter above the sinus of valsalva by pulsed A-mode ultrasonography. (b) The modified esophageal stethoscope is inserted and adjusted for optimal ultrasonic transmission and reception. Descending aortic blood velocity is continuously determined. (c) Cardiac output is determined by measuring with the suprasternal probe the ascending aortic load velocity. The monitor then calculates the constant proportionality factor by dividing the cardiac output by the descending aortic blood velocity. (d) The esophageal probe continually measures descending aortic blood velocity, and the monitor displays cardiac output as a function of the proportionality factor multiplied by the descending aortic blood velocity.



$$V = \frac{C \times Fd}{2F_0 \times \cos\theta}$$

where
- C = speed of sound
- Fd = frequency shift
- F_0 = frequency of the emitted sound
- θ = angle between the emitted sound and the moving object.

Recently, ultrasound probes have been incorporated into a variety of instruments that measure cardiac output. The main advantage of such devices, assuming their reasonable accuracy, is that they do not require intravascular placement. These devices also offer the possibility of continuous measurement of cardiac output.

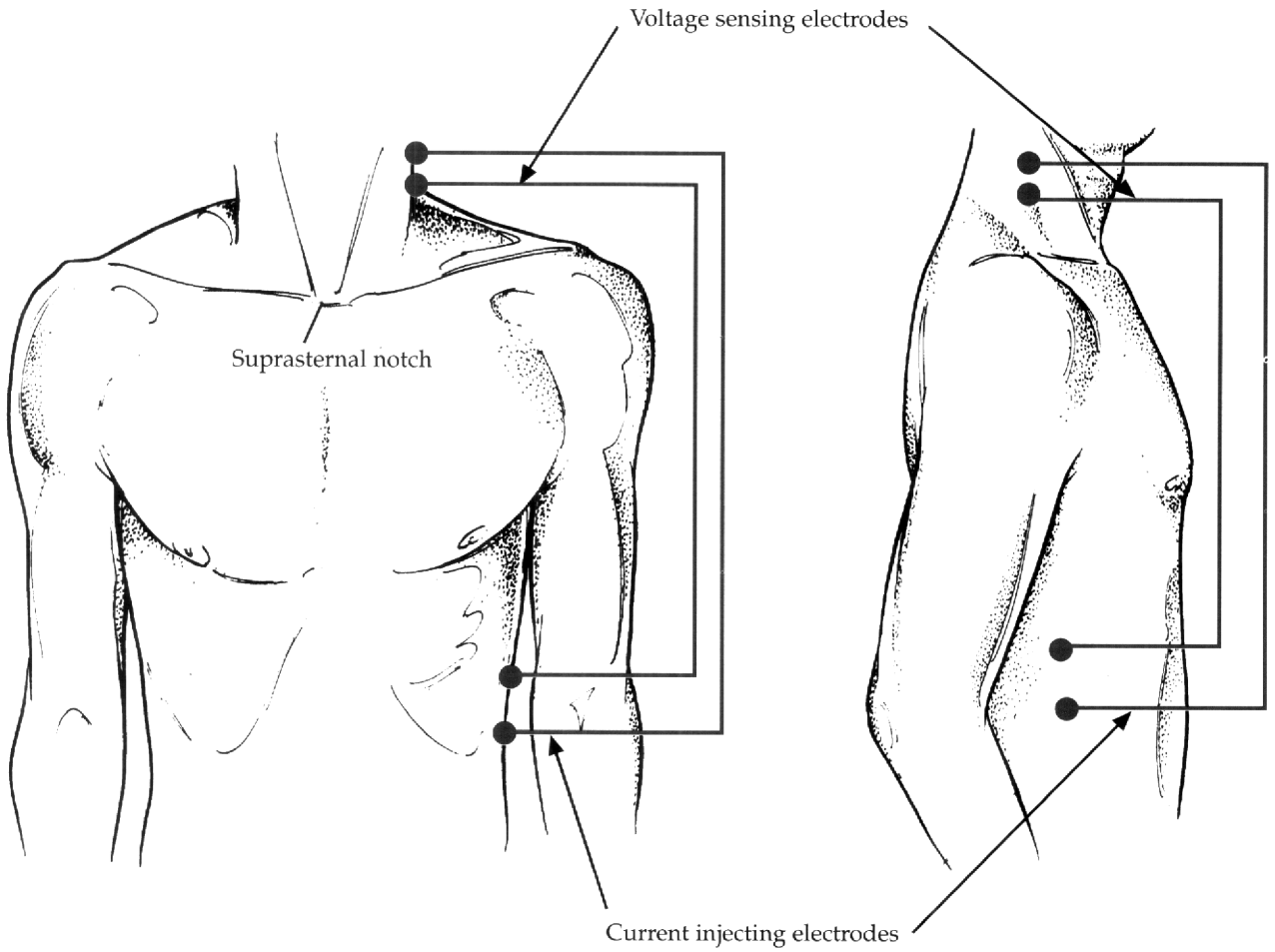
Blood velocity in the ascending aorta can be measured by a probe that is held in the operator's hand and positioned just above the sternal notch.[38] Blood velocity in the descending aorta can be measured by a probe attached to an esophageal stethoscope (Figure 4.2).[39, 40] A probe attached to an endotracheal tube can determine blood velocity in the pulmonary artery.[41, 42] When compared to standard thermodilution measurement of cardiac output, these instruments yield variable results, with correlation coefficients ranging from $r = 0.63$ to 0.91.[40, 43, 44] A variety of problems account for their relatively poor accuracy.

The cross-sectional area of the aorta (or pulmonary artery) can be measured by echo techniques but this is seldom practical in the clinical setting. Average human dimensions for the aorta are available from nomograms that consider sex, age, height, and weight, but individual patients may vary considerably from the average. Thus, the lack of precise cross-sectional area of the particular blood vessel remains a significant potential source of error.

In addition, the angle theta cannot be known precisely, which represents another source of error. Moreover, the ultrasound probe can move during use, causing theta to vary.

Because of problems with accuracy, determination of cardiac output by ultrasound has remained elusive. A recent attempt to overcome these problems employed a modified pulmonary artery catheter with three additional ultrasound transducers that measured pulmonary artery diameter and blood velocity.[45] The correlation be-

Figure 5.1 — Electrode placement for cardiac output measurement by thoracic bioimpedance.



tween ultrasound and thermodilution cardiac output with this instrument was r = 0.73. This device offers the advantage of continuous measurement of cardiac output by ultrasound during the intervals between thermodilution cardiac output determinations. Presumably, this instrument would allow calibration of ultrasound cardiac output using thermodilution cardiac output as the standard.

5.0 DETERMINATION OF CARDIAC OUTPUT BY BIOIMPEDANCE

In 1974, Kubicek and co-workers published a method for determining stroke volume by thoracic bioimpedance.[46] A complex concept, this technique is based upon the observation that resistance to a

current passed through the chest varies with thoracic aortic blood flow. Four pairs of surface electrocardiograph (ECG) electrodes are placed on the neck and chest (Figure 5.1). A constant, low amplitude, high-frequency alternative current is applied to two sets of electrodes, while the other two sets of electrodes are used to measure voltage changes. Kubicek and colleagues proposed the following equation to calculate stroke volume:

$$SV = \rho_b \times L^2/Z_0^2 \times T_{LVE} \times (dZ/dt)_{max}$$

where
- SV = stroke volume
- ρ_b = resistivity of blood (ohm x cm)
- L = electrical length of the thorax (cm)
- Z_0 = mean baseline impedance of the thorax (ohm)
- T_{LVE} = left ventricular ejection time (sec)
- $(dZ/dt)_{max}$ = maximum value of the first derivative of Z, where Z is the change in impedance caused by thoracic aortic blood flow.

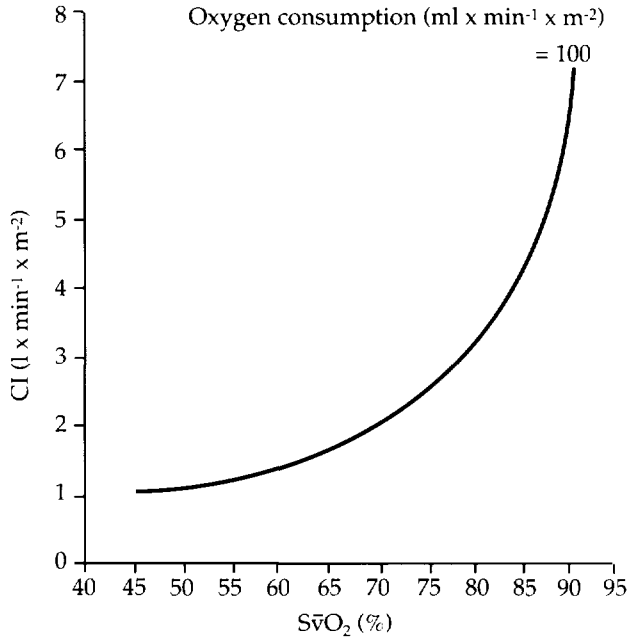
The equation developed by Kubicek and co-workers has been modified recently by Bernstein:[47]

$$SV = \frac{\delta \times (0.17H)^3 \times T_{LVE} \times (dZ/dt)_{max}}{4.2 \times Z_0}$$

where
- δ = correction factor for patient weight
- H = patient height (cm).

Application of the thoracic bioimpedance techniques has been disappointing because the correlation to thermodilution appears generally poor.[40] A completely noninvasive and continuous bioimpedance cardiac output monitor would be an attractive alternative to thermodilution, but only if the problems of accuracy can be solved.

Figure 6.1 — Calculated relationship between cardiac index (CI) and $S\bar{v}O_2$ at constant oxygen consumption and arterial oxygen content.



6.0 MIXED VENOUS OXYGEN SATURATION AND CARDIAC OUTPUT

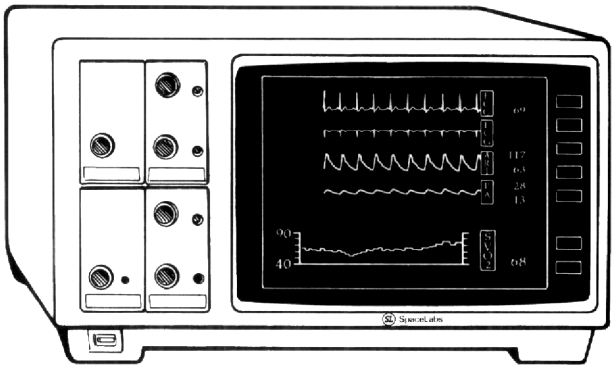
The Fick equation (see Section 1.2) states that cardiac output may be calculated by dividing total body oxygen consumption by the difference between arterial and mixed venous (pulmonary artery) oxygen content:

$$CO = \frac{O_2 \text{ consumption}}{(A\text{–}V\ O_2\Delta)}$$

The oxygen content of blood (either arterial or venous) is determined by the hemoglobin concentration, multiplied by the percentage of hemoglobin that is carrying oxygen (% saturation), multiplied by the oxygen carrying capacity of hemoglobin (1.34 ml O_2/g Hb), plus the volume of oxygen directly dissolved in blood (0.003 ml/mm Hg O_2 tension):

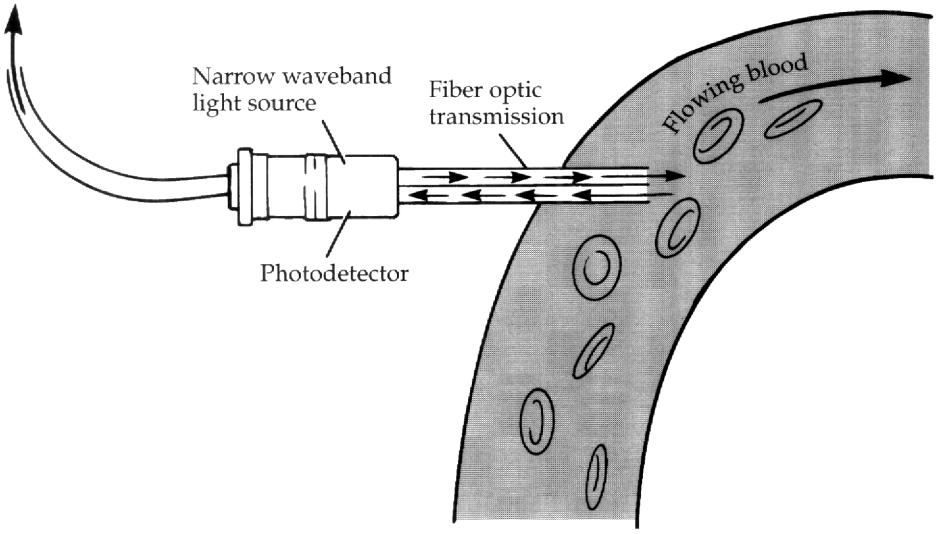
$$\text{Oxygen Content} = (1.34)(Hb)(SO_2) + (0.003)(PO_2)$$

Figure 6.2 — Reflection spectrophotometry, with in vivo fiber optic catheter, measuring light reflected by blood cells.

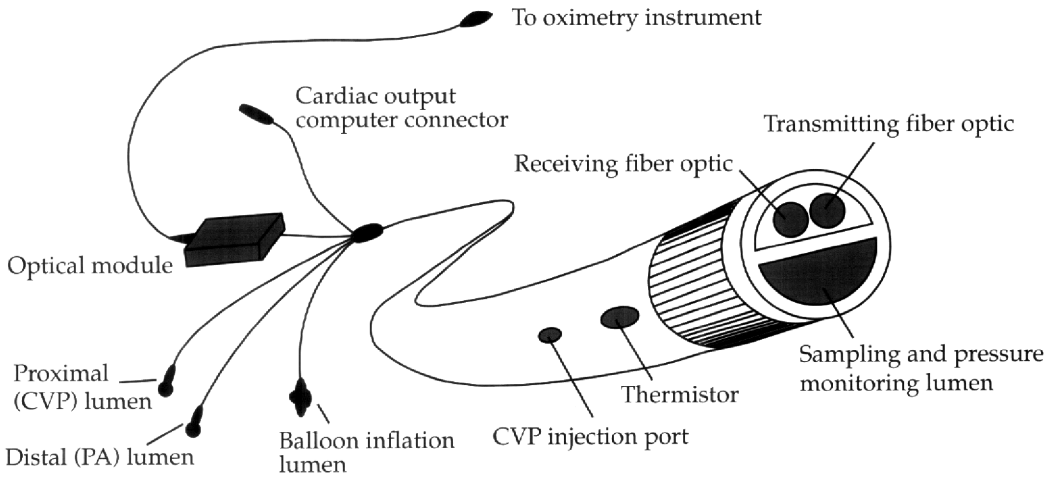


Processor

Output: oxyhemoglobin saturation ($S\bar{v}O_2$)



Fiber optic catheter



Normally, determination of cardiac output by the Fick method is impractical because of the need to measure all of the variables contained in the equation. However, the relationship between cardiac output and mixed venous oxygen saturation, described in the Fick equation, has clinical utility under certain circumstances. If oxygen consumption, arterial oxygen content, and hemoglobin remain constant for a period of time, then any change in cardiac output results in a change in mixed venous oxygen saturation, and the Fick equation can be simplified:

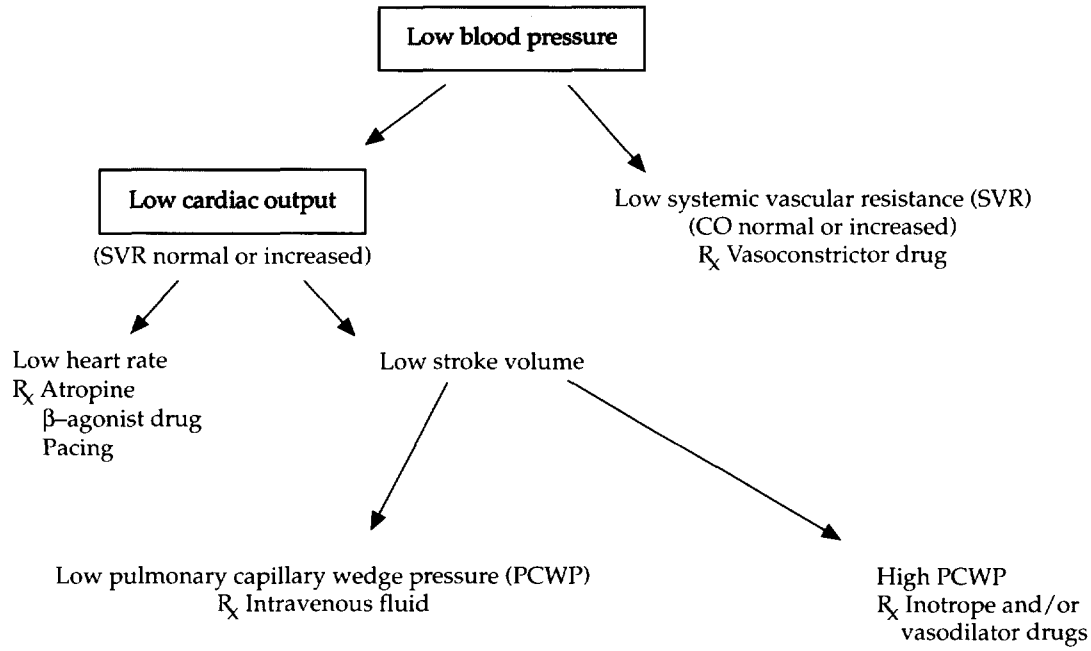
$$CO = \frac{X}{[Y - (Z)(S\bar{v}O_2)]}$$

where X, Y, and Z are held constant, and $S\bar{v}O_2$ equals mixed venous oxygen saturation.

Figure 6.1 illustrates the typical, non-linear relationship between $S\bar{v}O_2$ and cardiac output under conditions of constant oxygen consumption, arterial oxygen content, and hemoglobin. As cardiac output declines, the supply of oxygen to tissues declines also. Tissues then extract a greater percentage of the available oxygen from the blood, leaving relatively less oxygen in the venous blood returning to the heart. This is detected as a fall in mixed venous oxygen saturation, which is normally in the range of 60-80%. When mixed venous oxygen saturation falls below 50%, the supply of oxygen to tissues has generally become inadequate.

The technology of reflection spectrophotometry allows mixed venous oxygen saturation to be measured and displayed continuously by the use of a fiber optic detection system incorporated into a pulmonary artery catheter (Figure 6.2). A change in $S\bar{v}O_2$ serves to alert the clinician to a change in the status of the patient. The change in $S\bar{v}O_2$ may be due to a change in cardiac output, if oxygen consumption, arterial oxygen content, and hemoglobin are constant (Figure 6.1); under these circumstances the $S\bar{v}O_2$ monitor may be used to follow trends in cardiac output. However, in critically ill patients, oxygen consumption, arterial oxygen content, or hemoglobin may change at any time. Therefore, cardiac output, arterial oxygen saturation, and hemoglobin should be measured before ascribing a major change in $S\bar{v}O_2$ to any particular cause.

Figure 7.1 — This branching diagram illustrates the differential diagnosis of hypotension and low cardiac output syndromes, using a pulmonary artery catheter with thermodilution cardiac output capability.



7.0 CLINICAL INTERPRETATION OF CARDIAC OUTPUT

Blood pressure is the hemodynamic variable most frequently used to assess overall cardiovascular performance. Readily measured by noninvasive methods, blood pressure determination is clinically significant because blood flow to tissues can be inadequate when blood pressure is too low (a systolic pressure below 90 mm Hg or a mean blood pressure below 60 mm Hg is usually considered to be abnormal in an adult individual). However, a normal blood pressure does not always indicate optimal blood flow to the tissues. The volume of blood flow, hence the quantity of oxygen and other vital substrates that are delivered to tissues, must also be considered. Cardiac output serves as the variable that describes the total volume of blood flow in the circulation per unit time.

Cardiac output and blood pressure are related by the equation:

$$CO = \frac{(MBP - CVP) \times 80}{SVR}$$

where
- CO = cardiac output
- MBP = mean blood pressure
- CVP = central venous pressure
- SVR = systemic vascular resistance (calculated after measuring the other variables)
- 80 = proportionality constant related to units of measurement.

This equation is analogous to the equation in electrical physics:

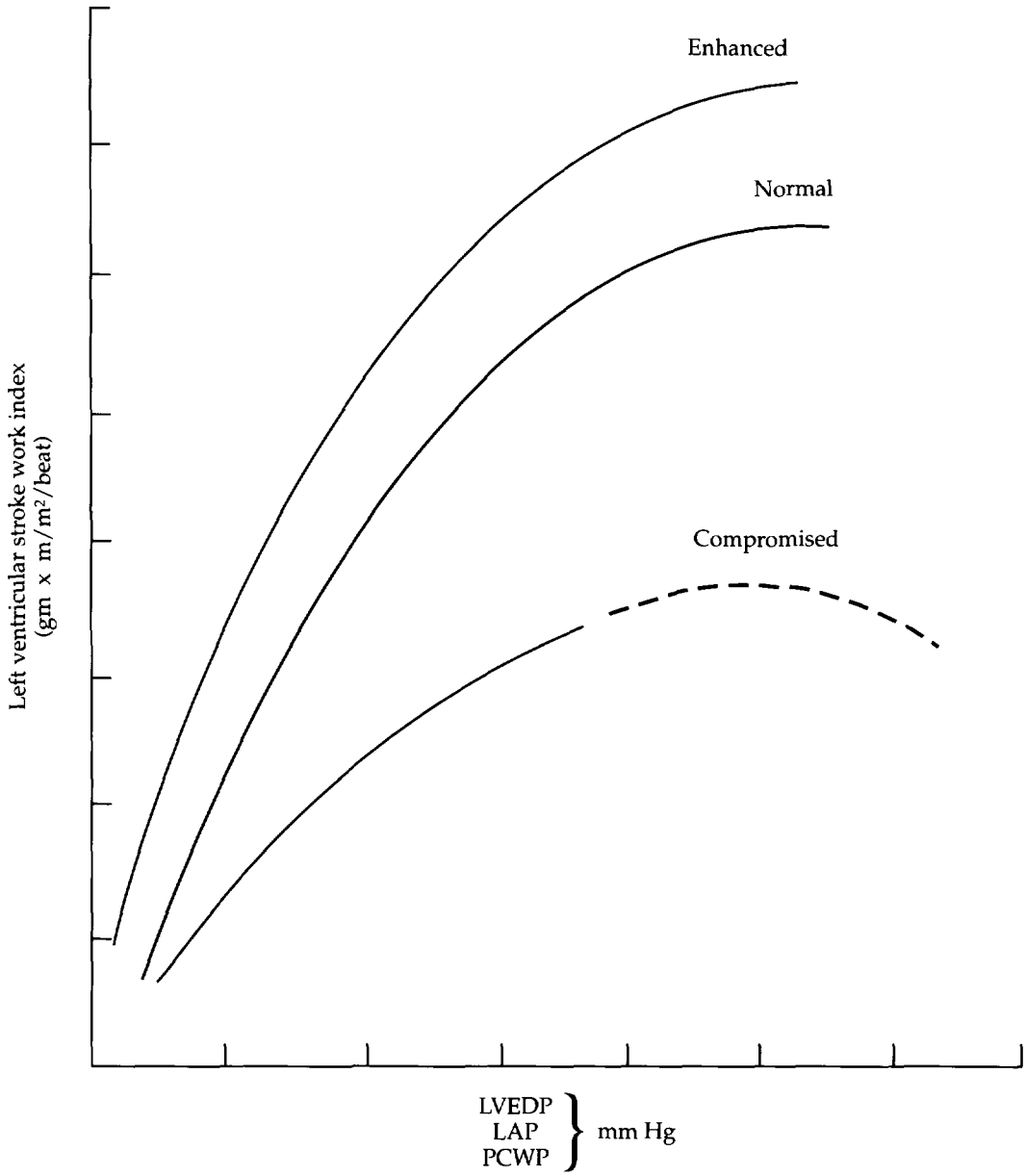
$$I = \frac{E}{R}$$

where
- I = current (analogous to CO)
- E = voltage drop in the circuit (analogous to the pressure drop between the aorta and the right atrium, MBP – CVP)
- R = resistance (analogous to SVR).

Hypotension is a common problem in critical care medicine and can be divided into two major categories, based upon whether the hypotension is due primarily to low cardiac output or low resistance (SVR). The therapy for this condition differs depending upon the cause. If cardiac output is low, as in patients with cardiac failure or hemorrhagic shock, treatment focuses on improving cardiac output. If cardiac output is normal or elevated and resistance is low, as in bacterial sepsis or an allergic reaction, the therapy aims to increase the SVR by pharmacologic means (Figure 7.1). The ability to measure cardiac output as well as blood pressure enables the clinician to manipulate the abnormal cardiovascular system in a rational fashion to improve blood flow to tissues.

The cardiac output is also useful for understanding and manipulating the pumping function of the heart. The Frank-Starling mechanism defines a characteristic relationship between the volume of blood filling the left ventricle (end-diastolic volume) and the

Figure 7.2 — The Frank-Starling relationship between left ventricular stroke work and left ventricular end-diastolic volume is illustrated for hearts with normal function, depressed function, and function enhanced by inotropic drugs. In the clinical setting, left ventricular stroke work (LVSW) is often replaced by cardiac output (CO), stroke volume (SV), or ejection fraction (EF). End-diastolic volume is replaced by left atrial pressure (LAP) or pulmonary artery wedge or occlusion pressure (PCWP).



amount of blood pumped by the heart during each contraction (left ventricular stroke volume) (Figure 7.2). As the blood volume in the left ventricle increases, the cardiac muscle stretches, develops greater mechanical energy during contraction, and ejects greater volumes of blood. However, if the ventricle becomes too large, the heart muscle overdistends and stroke volume actually declines. Ventricular filling volume is usually determined by measuring filling pressure in the clinical setting, since ventricular filling volume and filling pressure are closely related. The pulmonary artery wedge pressure, measured with a pulmonary artery catheter, serves as a close approximation of left ventricular end diastolic pressure. The ventricular filling pressure (and volume) can be manipulated by administering appropriate drugs and fluids, to optimize cardiac output and treat various cardiovascular abnormalities.

The condition of low cardiac output is another common problem in critical care medicine that can be divided into two major categories, based upon whether pulmonary artery wedge pressure is high or low. If pulmonary artery wedge pressure is low, the first step in improving cardiac output is to administer intravenous fluids to increase the left ventricular filling volume. If pulmonary artery wedge pressure is too high, the cardiac output is low because the ventricle is failing to contract normally. This problem can be treated by administering drugs (inotropes) to improve contractility. If the ventricle is overdistended, vasodilating drugs can also be used to lower left ventricular filling volume to a point where the Frank-Starling mechanism results in optimal cardiac output.

8.0 METHODOLOGY FOR PLACEMENT OF THE PULMONARY ARTERY CATHETER

Placement of a pulmonary artery catheter is ordinarily a relatively simple procedure that can be accomplished in 10 to 20 minutes by an experienced practitioner. Catheter insertion can be divided into two stages: gaining intravascular access and then floating the pulmonary artery catheter into the pulmonary artery.

Intravascular access is usually obtained by the Seldinger technique in which a needle is inserted into an appropriate vein, first

Figure 8.1 — (a) The introducer sheath is placed over the guidewire that was previously inserted into the vein. (b) The introducer sheath is advanced over the guide wire, through the skin, and into the vein. (c) The guide wire is removed, leaving the introducer sheath in the vein. (d) The pulmonary artery catheter is advanced into the vein through the introducer sheath.

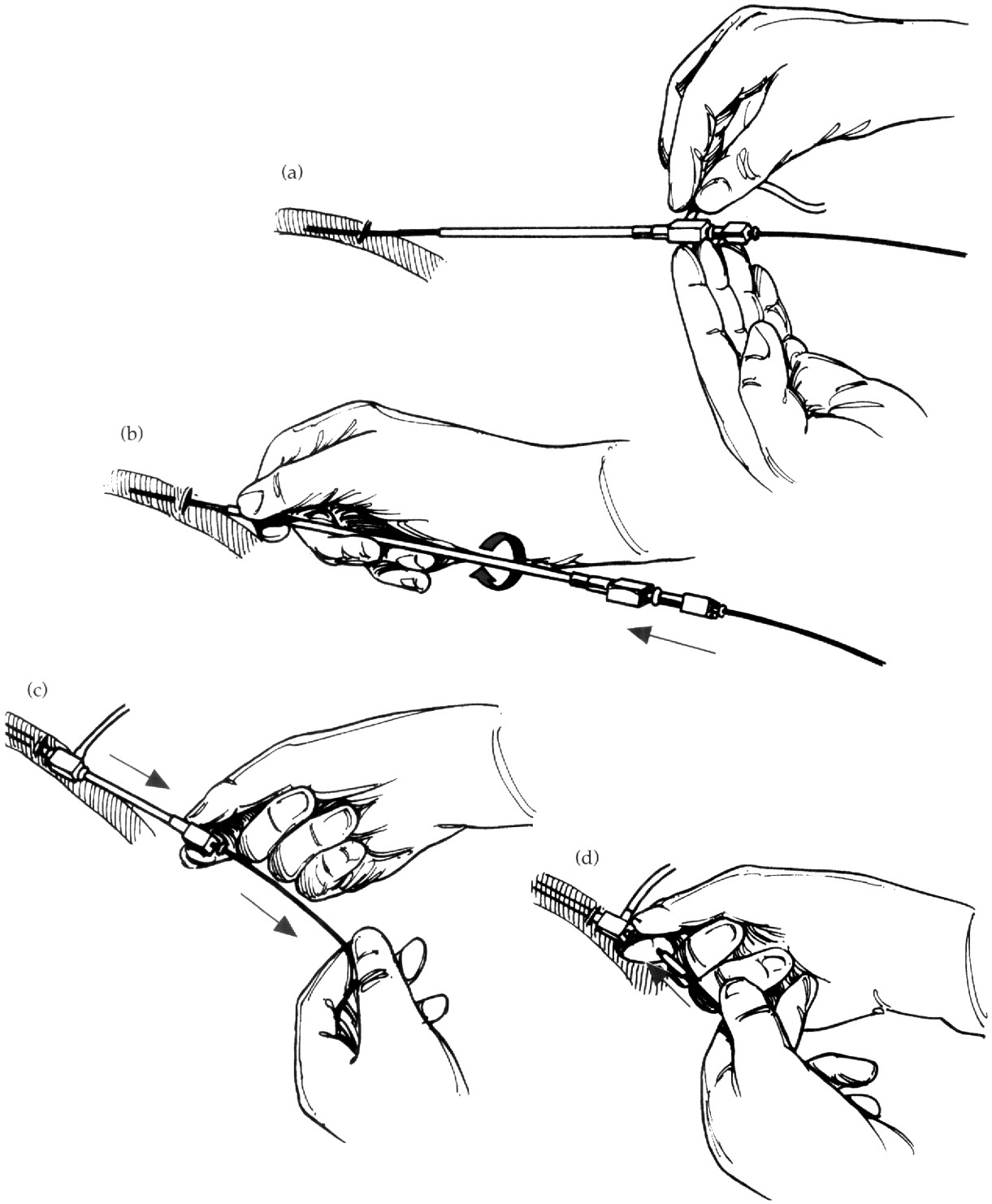


Figure 8.2 — The major veins of the upper body, suitable for placing a pulmonary artery catheter: The internal jugular vein and subclavian vein are used most often because of their large size and proximity to the heart.

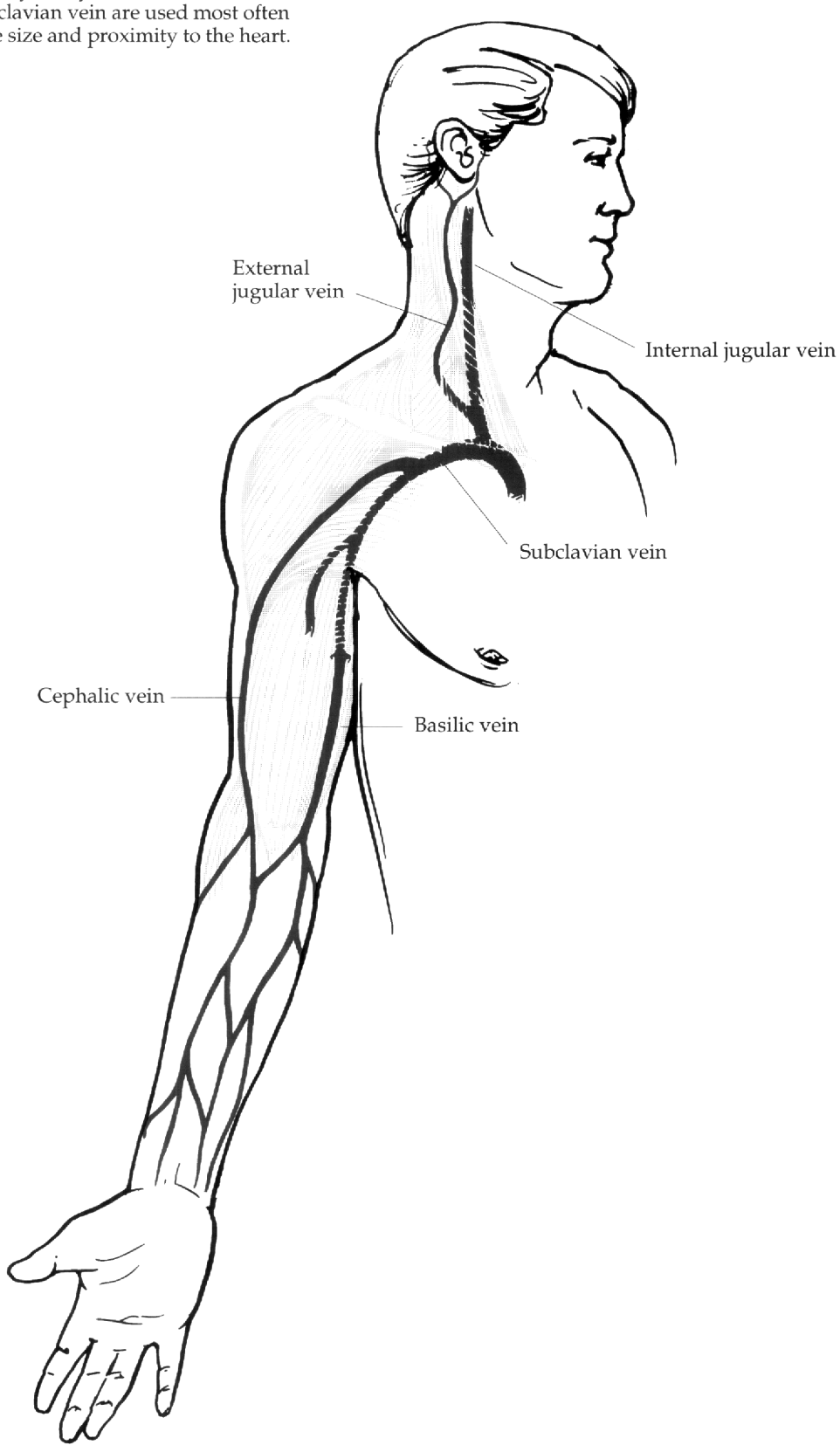
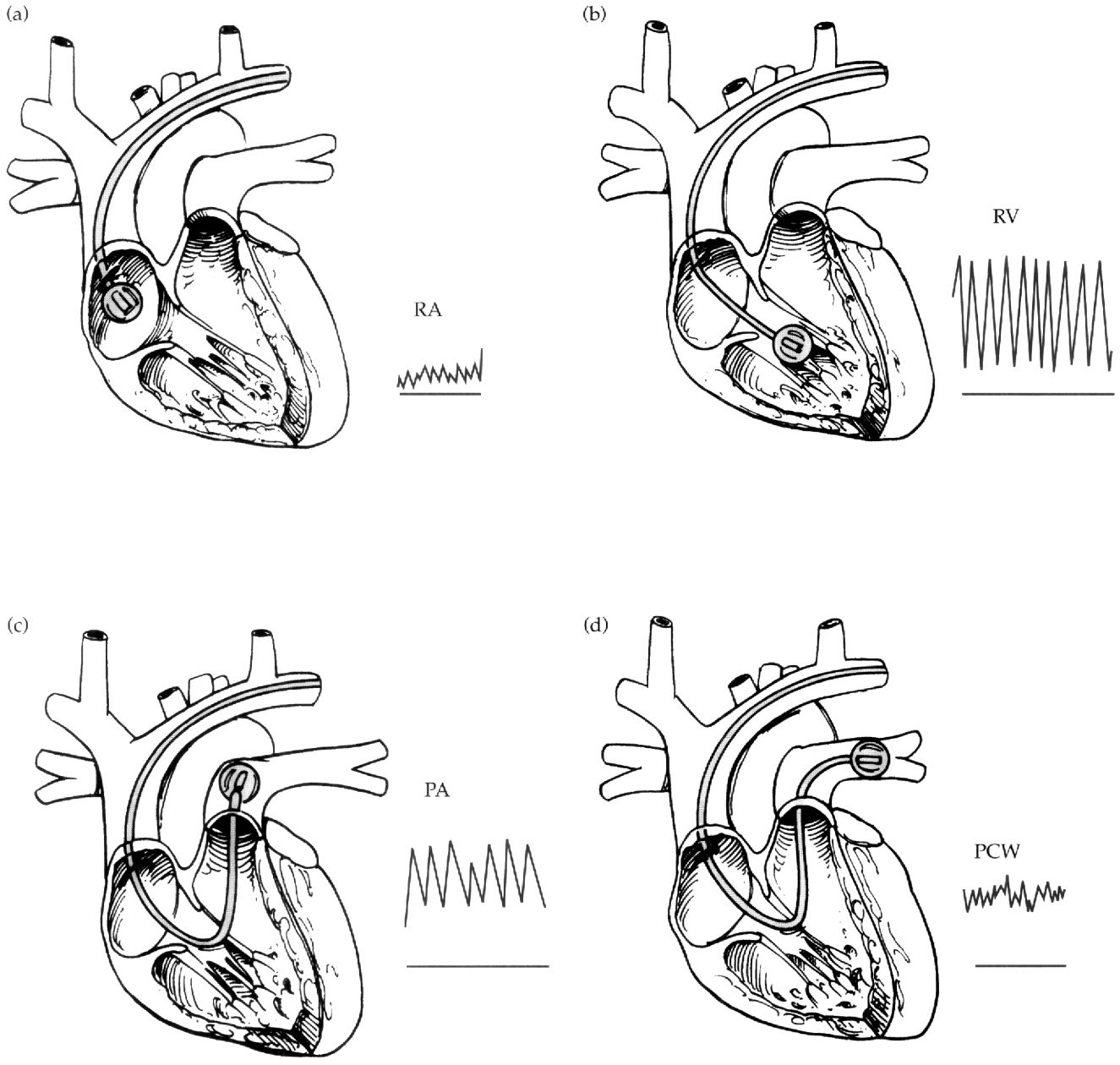


Figure 8.3 — The characteristic pressure waveforms recorded from the distal port of the pulmonary artery catheter: The catheter passes from right atrium, to right ventricle, to pulmonary artery, and to pulmonary artery wedge position.



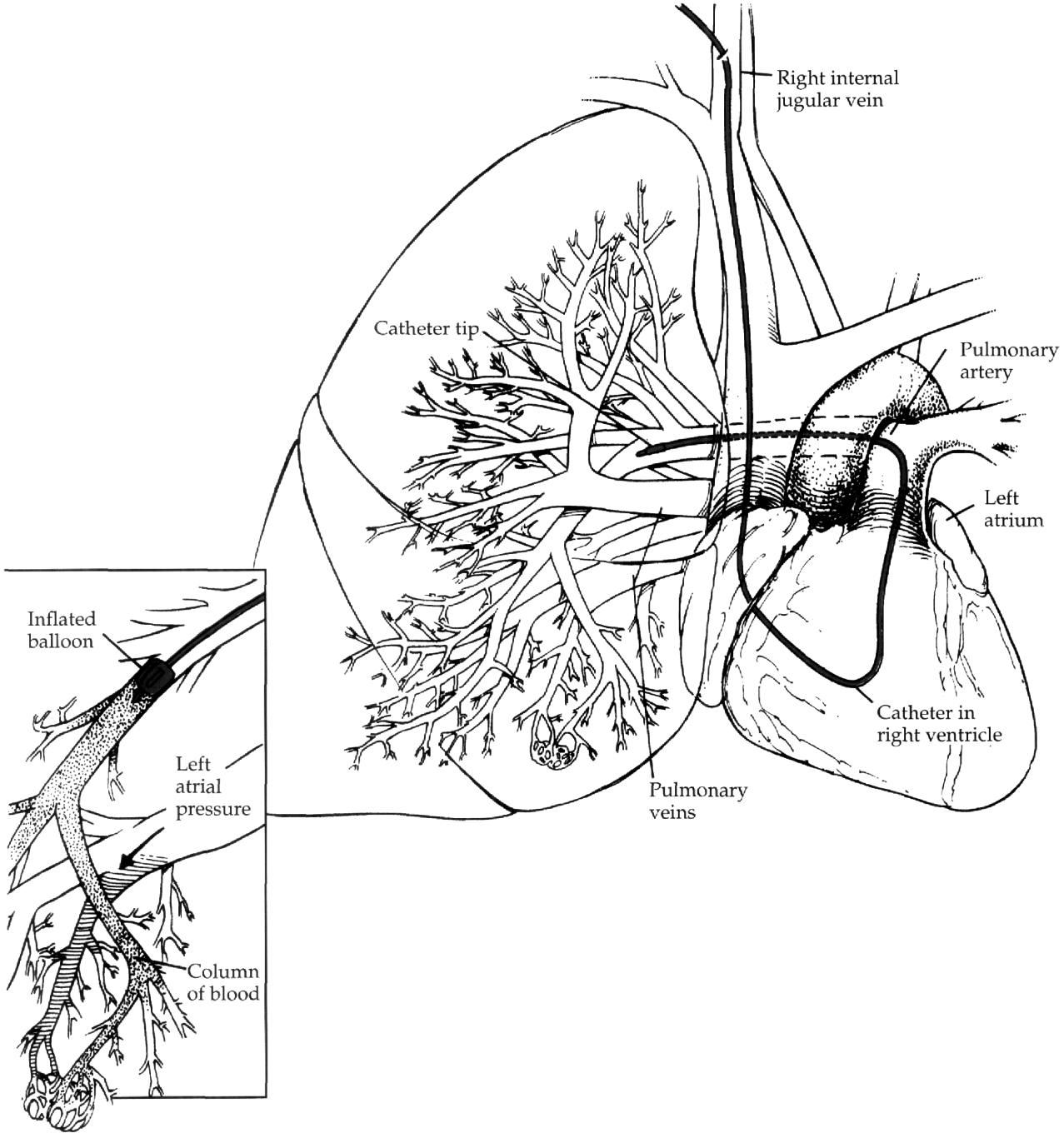
identified by the aspiration of blood into a syringe attached to the needle (Figure 8.1). A flexible wire is then threaded through the needle and advanced well into the vein. After removing the needle from the wire, a pulmonary artery catheter introducer sheath is placed over the wire and inserted into the vein, using the wire as a guide. The introducer sheath serves as a conduit for the pulmonary artery catheter.

Any superficial vein large enough to accommodate the introducer sheath could theoretically be used for placement of the pulmonary artery catheter. However, the large veins of the upper body, either the internal jugular veins of the neck or the subclavian veins, which are located just below each clavicle, are usually used for practical reasons (Figure 8.2). Under unusual circumstances the basilic vein of the arm or the femoral vein in the groin can be used as well.

Once the introducer sheath has been situated, the pulmonary artery catheter is inserted through the introducer sheath and advanced until the tip of the catheter reaches the right atrium. This point is identified by determining the typical distance from the introducer sheath to the right atrium and by the appearance of a right atrial pressure tracing from the distal port of the pulmonary artery catheter. The balloon of the pulmonary artery catheter is then inflated and the catheter is advanced. The balloon floats the catheter tip into the stream of blood as it flows into the right ventricle, through the pulmonic valve and into the pulmonary artery. The location of the catheter can be determined at any time by the characteristic appearance of the pressure tracing from the right ventricle or pulmonary artery (Figure 8.3). After entering the pulmonary artery, the catheter is advanced slowly with the balloon inflated until the balloon wedges in the artery and the characteristic pulmonary artery wedge pressure (or occlusion pressure) tracing is obtained. This is the downstream pressure, beyond the inflated balloon, which ordinarily reflects left ventricular filling pressure (Figure 8.4).

Placement of the introducer sheath and the pulmonary artery catheter is relatively safe but entails certain risks. Complications comprise those associated with placement of the introducer sheath and those associated with the pulmonary artery catheter. Complications associated with obtaining vascular access include pneumo-

Figure 8.4 — The pulmonary artery catheter with its relationships to the heart, lung, and pulmonary vessels: When the balloon is inflated to occlude the pulmonary artery, the distal port of the catheter measures the venous backpressure downstream from the balloon. This pressure ordinarily reflects left atrial pressure, as shown in the inset diagram.



thorax and needle puncture of nearby arteries, such as the carotid or subclavian arteries. Pneumothorax typically requires evacuation by placement of a chest tube. Complications associated with the pulmonary artery catheter include ventricular arrhythmias, such as premature ventricular contractions (PVCs), bundle branch block, complete heart block, pulmonary infarction, perforation of the heart wall, and perforation of the pulmonary artery. In a report of 6,245 cases, Shah and co-workers noted the following incidence of complications: PVCs requiring therapy (3.1%), right bundle branch block (0.048%), left bundle branch block (0.016%), complete heart block (0.016%), perforation of the pulmonary artery (0.08%), minor pulmonary infarcts (0.064%), and perforation of the right ventricle (0.016%).[48] Perforation of the pulmonary artery is generally the most serious complication and resulted in one death in the series reported by Shah. The other complications listed are not ordinarily fatal. The overall incidence of complications from placement of pulmonary artery catheters is small enough that the benefits of catheterization appear to exceed the risks in appropriate cases.

9.0 REFERENCES

1. Rushmer R: The cardiac output. In: Cardiovascular Dynamics. Philadelphia: WB Saunders, 1961.
2. Guyton AC, Jones CE, Coleman TG: Normal cardiac output and its variations. In: Circulatory Physiology: Cardiac Output and Its Regulation. Philadelphia: WB Saunders, 1973.
3. Rowell LB: Human Circulation, Regulation During Physical Stress. New York: Oxford University Press, 1986.
4. Fick A: Uber die Messung des Blutquantums in den Herzen Ventrikeln. Würzburg, Sitz. Phys-Med Ges, July 9, 1870.
5. Patton HD, Fuchs AF, Hille B, et al (Eds): Textbook of Physiology. Philadelphia: WB Saunders, 1989.
6. Taylor BC, Sheffer DB: Understanding techniques for measuring cardiac output. Biomed Instrument Tech 3:188-197, 1990.
7. Reddy PS, Curtiss EJ, Bell B, et al: Determination of cardiac output during diagnostic catheterization. Fick vs dye cardiac output. J Lab Clin Med 87: 568-576, 1976.
8. Stewart GN: Researches on the circulation time and on the influences which affect it. IV - The output of the heart. J Physiol 22:159, 1897.
9. Hamilton WF, Moore JW, Kinsman JM, et al: Studies on the circulation. Am J Physiol 99:534-555, 1932.
10. Newman EV, Merrell M, Genecin A, et al: The dye dilution method of describing the central circulation. Circulation 4:735-746, 1951.
11. Hillis LD, Firth BC, Williford MD: Comparison of thermodilution and indocyanine green dye in low cardiac output or left-side regurgitation. Am J Cardiol 57:1201-1202, 1986.
12. Ganz W, Donoso R, Marcus H: A new technique for measurement of cardiac output by thermodilution in man. Am J Cardiol 27:392-396, 1971.
13. Forrester JS, Ganz W, Diamond G, et al: Thermodilution cardiac output determination with a single flow-directed catheter. Am Heart J 83: 306-311, 1972.
14. Kaplan JA (Ed): Cardiac Anesthesia (Volume 1). Philadelphia: WB Saunders, 1987.
15. Stewart GN: The output of the heart in dogs. Am J Physiol 57:27-50, 1921.
16. Moore JW, Kinsman JM, Hamilton WF, et al: Studies on the circulation. II. Cardiac output determination; comparison of the injection method with the direct Fick procedure. Am J Physiol 89:331-339, 1929.
17. Kodata LT: Theory and application of thermodilution cardiac output measurement: a review. Heart Lung 14:605-614, 1985.
18. Ganz W, Swan HJC: Measurement of blood flow by thermodilution. Am J Cardiol 29:241-246, 1972.
19. Yelderman ML, Quinn MD, McKown RC: Thermal safety of a filamented pulmonary artery catheter. J Clin Monit 8:147-149, 1992.
20. Yelderman M: Continuous measurement of cardiac output with the use of stochastic system identification techniques. J Clin Monit 6:322-332, 1990.
21. Yelderman ML, Ramsay MA, Quinn MD, et al: Continuous thermodilution cardiac output measurement in intensive care unit patients. J Cardiothorac Vasc Anesth 6:270-274, 1992.
22. Rapaport E: Usefulness and limitations of thermal washout techniques in ventricular volume measurement. Am J Cardiol 18:226-234, 1966.
23. Hurford WE, Zapol WM: The right ventricle and critical illness: a review of anatomy, physiology, and clinical evaluation of its function. Intensive Care Med 14:448-457, 1988.
24. Levett JM, Replogle RL: Thermodilution cardiac output: a critical analysis and review of the literature. J Surg Res 27:392-404, 1979.
25. Skoda A, Rowe GG, Cesar AC, et al: Intravascular and intracardiac blood temperatures in man. J Appl Physiol 17:706-708, 1962.
26. Wessel HU, James GW, Paul MH: Effects of respiration and circulation on central blood temperature of the dog. Am J Physiol 211: 1403-1412, 1966.
27. Elkayam U, Berkley R, Stanley A, et al: Cardiac output by thermodilution technique. Effect of injectate's volume and temperature on accuracy and reproducibility in the critically ill patient. Chest 84:418-422, 1983.
28. Armengol J, Man GCW, Balsys AJ, et al: Effects of the respiratory cycle on cardiac output measurements: reproducibility of data enhancement by timing the thermodilution injections in dogs. Crit Care Med 9:852-854, 1981.
29. Stevens JH, Raffin TA, Mihm FG, et al: Thermodilution cardiac output measurement. Effects of the respiratory cycle on its reproducibility. JAMA 253:2240-2242, 1985.
30. Snyder JV, Powner DJ: Effects of mechanical ventilation on the measurement of cardiac output by thermodilution. Crit Care Med 10:677-682, 1982.

31. Meisner H, Glanert S, Steckmeier B, et al: Indicator loss during injection in the thermodilution system. Res Exp Med 159:183-196, 1973.
32. Wessel HU, Paul MH, James GW, et al: Limitations of thermal dilution curves for cardiac output determinations. J Appl Physiol 30:643-652, 1971.
33. Pavek K, Boska E, Selecky FV: Measurement of cardiac output by thermodilution with constant rate injection of indicator. Circ Res 15:311, 1964.
34. Sorensen MB, Bille-Brahe NE, Engell HC: Cardiac output measurement by thermal dilution: reproducibility and comparison with the dye-dilution technique. Ann Surg 183:67-72, 1976.
35. Nara AR, Burns MP, Downs WG: Biophysical Measurement Series: Blood Pressure. Redmond: SpaceLabs Medical, Inc., 1989.
36. Wetzel RC, Latson TW: Major errors in thermodilution measurement of cardiac output caused by rapid volume infusion. Anesthesiology 61:A90, 1984.
37. Fischer AP, Benis AM, Jurado RA, et al: Analysis of errors in measurement of cardiac output by simultaneous dye and thermal dilution in cardiothoracic surgical patients. Cardiovas Res 12:190-199, 1978.
38. Huntsman LL, Stewart DK, Banes DR, et al: Noninvasive Doppler determinations of cardiac output in man. Circulation 67:593-602, 1983.
39. Mark J, Steinbrook R, Gugino L, et al: Continuous noninvasive monitoring of cardiac output with esophageal Doppler ultrasound during cardiac surgery. Anesthsiol Anal 65:1013-1020, 1986.
40. Siegel LC, Shafer SL, Martinez G, et al: Simultaneous measurements of cardiac output by thermodilution esophageal Doppler and electrical impedance in anesthetized patients. J Cardiothorac Anesthsiol 2:590-595, 1988.
41. Abrams J, Weber RE, Holman K: Transtracheal Doppler: a new procedure for continuous cardiac output. Anesthesiology 70:134-138, 1989.
42. Abrams J, Weber RE, Holman KD: Continuous cardiac output determination using transtracheal Doppler: initial results in humans. Anesthesiology 71:11-15, 1989.
43. Lamantia K, Barash P: Cardiac output: measurement of the future or of the past. Cardiothoracic Anesthsiol 2:587-589, 1988.
44. Wong DH, Tremper KK, Stemmer EA, et al: Noninvasive cardiac output: simultaneous comparison of two different methods with thermodilution. Anesthesiology 72:784-792, 1990.
45. Allard MW, Robinson LM, Leone BJ: The continuous determination of cardiac output using a flow directed Doppler pulmonary artery catheter. Society of Cardiovascular Anesthesiologists, Annual Meeting Abstracts, p. 207, 1990.
46. Kubicek WG, Kottke FJ, Ramos MU, et al: The Minnesota impedance cardiograph - theory and applications. Biomed Engineer 9:410-416, 1974.
47. Bernstein DP: A new stroke volume equation for thoracic electrical bioimpedance: theory and rationale. Crit Care Med 14:904-909, 1986.
48. Shah KB, Rao TLK, Laughlin S: A review of pulmonary artery catheterization in 6,245 patients. Anesthesiology 61:271-275, 1984.

10.0 ILLUSTRATION CREDITS

Figure 1.1
Rowell L: Human Circulation Regulation During Physical Stress. New York, Oxford University Press, 1986.

Figure 1.2
Netter F: Nervous System (Part 1). West Caldwell, New Jersey, Ciba Pharmaceutical, 1983.

Figure 1.3
Taylor BC, Sheffer DB: Understanding techniques for measuring cardiac output. Biomed Instrument Technol: May-June, 1990.

Figure 2.10
Dhainut JF, Brunet F, Monsallier JF, et al: Bedside evaluation of right ventricular performance using a rapid computerized thermodilution method. Crit Care Med 15: 148-152, 1987.

Figure 3.1
Stetz CW, Miller RG, Kelly GE, Raffin TA: Reliability of the thermodilution method in the determination of cardiac output in clinical practice. Am Rev Respir Dis 126:1001-1004, 1982.

Figure 3.2
Afonso S, Rowe GR, Castillo CA, Crumpton CW: Intravascular and intracardiac blood temperatures in man. J Appl Physiol 17:706-708, 1962.

Figure 3.4
Levett JM, Replogle RL: Thermodilution cardiac output: a critical analysis and review of the literature. J Surg Res 27:392-404, 1979.

Figure 3.5
Wetzel RC, Latson TW: Major errors in thermodilution measurement of cardiac output caused by rapid volume infusion. Anesthesiology 61:A90, 1984.

Figure 3.6
Fisher AP, Benis AM, Jurado RA, et al: Analysis of errors in measurement of cardiac output by simultaneous dye and thermal dilution in cardiothoracic surgical patients. Cardiovas Res 12:190-199, 1978.

Figure 4.1
Scher AM: Textbook of Physiology. Philadelphia, WB Saunders, 1989.

Figure 4.2a
Abrams J, Weber RE, Holman KD: Continuous cardiac output determination using transtracheal Doppler: Initial results in humans. Anesthesiology 71:11-15, 1989.

Figure 4.2b,c,d
Freund PR: Transesophageal Doppler scanning versus thermodilution during general anesthesia. Am J Surg 153:490-494, 1989.

Figure 5.1
Wong, DH, Tremper KK, Stemmer EA: Noninvasive cardiac output: simultaneous comparison of two different methods with thermodilution. Anesthesiology 72: 784-792, 1990.

Figure 6.1
Gravenstein JS, Paulus DA: Clinical Monitoring Practice. Philadelphia, JB Lippincott Company, 1987.

Figure 6.2
Fahey PJ (Ed): Continuous Measurement of Blood Oxygen Saturation in the High Risk Patient. San Diego, Beach International, Inc., 1985.

Figure 7.2
Reeves JG: Vasoactive Drugs and When to Use Them. Manual of Cardiac Anesthesia. New York, Churchill Livingstone, Inc., 1984.

Figure 8.1
Urbach DR, Rippe JM: Intensive Care Medicine. Boston, Little Brown and Company, 1985.

Figure 8.2
Seneff MG, Rippe JM: Intensive Care Medicine. Boston, Little Brown and Company, 1985.

Figure 8.3
American Edwards Laboratories: Understanding Hemodynamic Measurements Made with the Swan-Ganz® Catheter. Anaheim, published by American Edwards Laboratories, Division of American Hospital Supply Corp, September 1980.
Swan-Ganz® is a registered trademark of Baxter International, Inc.

Figure 8.4
Abrams JH, Cerra F, Holcroft JW: Care of the Surgical Patient. New York, Scientific American, Inc., 1989.

11.0 BIBLIOGRAPHY

The following bibliography offers a chronological listing of citations pertinent to the study and determination of cardiac output measurement.

11.1 Thermodilution Cardiac Output Measurement

Hamilton WF, Riley RL, Attyah AM, et al: Comparison of the Fick and dye injection methods of measuring cardiac output in man. Am J Physiol 153:309-321, 1948.

Visscher MB, Johnson JA: The Fick principle: analysis of potential errors in its conventional application. J Appl Physiol 5:635-638, 1953.

Selzer A, Sudrann RB: Reliability of the determination of cardiac output in man by means of the Fick principle. Circ Res 6:485-490, 1958.

Afonso S, Herrick JF, Youmans WB, et al: Temperature variations in the venous system of dogs. Am J Physiol 203:278-282, 1962.

Zierler KL, Nichols RJ, Traber DL: Theoretical basis of indicator dilution methods for measuring flow and volume. Circ Res 10:393-407, 1962.

Hosie KF, Husserl FE: Thermodilution techniques. Circ Res 10:491-504, 1962.

Rapaport E, Wiegand BD, Bristow JD, et al: Estimation of left ventricular residual volume in the dog by a thermodilution method. Circ Res 11:803-810, 1962.

Bristow JD, Ferguson RE, Mintz F, et al: Thermodilution studies of ventricular volume changes due to isoproterenol and bleeding. J Appl Physiol 18:129-133, 1963.

Opdyke DF: Agreement of cardiac outputs calculated from paired indicator dilution curves. J Appl Physiol 20:9-15, 1965.

Olsson B, Pool J, Vandermoten P, et al: Validity and reproducibility of determination of cardiac output by thermodilution in man. Cardiology 55:136-148, 1970.

Ganz W, Donoso R, Marcus HS, et al: A new technique for the measurement of cardiac output by thermodilution in man. Am J Cardiol 27:392-396, 1971.

Sanmarco ME, Philips CM, Marquez LA, et al: Measurement of cardiac output by thermodilution. Am J Cardiol 28:54-58, 1971.

Lipp H, O'Donoghue K, Resnekov L: Intracardiac knotting of a flow-directed balloon catheter (letter). N Engl J Med 19:220, 1971.

Forrester JS, Ganz W, Diamond G, et al: Thermodilution cardiac output determination with a single flow-directed catheter. Am Heart J 83:306-311, 1972.

Ganz W, Swan HJC: Measurement of blood flow by thermodilution. Am J Cardiol 29:241-246, 1972.

Vliers A, Oesoburg B, Visser KR, Zijlstra WG: Choice of detection site for the determination of cardiac output by thermodilution: the injection thermistor catheter. Cardiovas Res 7:133-138, 1973.

Meisner H, Hagl S, Heimisch W, et al: Evaluation of the thermodilution method of measurement of cardiac output after open heart surgery. Ann Thoracic Surg 18:504-515, 1974.

Hodges M, Downs JB, Mitchell LA: Thermodilution and Fick cardiac index determinations following cardiac surgery. Crit Care Med 3:182-184, 1975.

Weisel RD, Berger RL, Hechtman HB: Measurement of cardiac output by thermodilution. N Engl J Med 292:682-684, 1975.

Wyse SD, Pfitzner J, Rees A, et al: Measurement of cardiac output by thermodilution in infants and children. Thorax 30:262-265, 1975.

Berger RL, Weisel RD, Vito L, et al: Cardiac output measurement by thermodilution during cardiac operations. Ann Thoracic Surg 21:43-47, 1976.

Forrester JS, Diamond G, Chatterjee K, Swan HJC: Medical therapy of acute myocardial infarction by application of hemodynamic subsets (First of two parts). N Engl J Med 295:1356-1362, 1976.

Mathur M, Harris EA, Yarrow S, et al: Measurement of cardiac output by thermodilution in infants and children after open heart operations. J Thoracic Cardiovas Surg 72:221-225, 1976.

Reddy PS, Curtiss EI, Bell B, et al: Determinants of variation between Fick and indicator dilution estimates of cardiac output during diagnostic catheterization. Fick vs dye cardiac outputs. J Lab Clin Med 87:568-576, 1976.

Snoeckx LH, Verheyen JL, Van de Water A, et al: On-line computation of cardiac output with the thermodilution method using a digital minicomputer. Cardiovas Res 10:556-564, 1976.

Woods M, Scott RN, Harken AH: Practical considerations for the use of a pulmonary artery thermistor catheter. Surgery 79:469-475, 1976.

Dizon CT, Gezari WA, Barash PG, Crittenden JF: Hand held thermodilution cardiac output injector. Crit Care Med 5:210-212, 1977.

Forrester JS, Diamond GA, Swan HJC: Correlative classification of clinical and hemodynamic function after acute myocardial infarction. Am J Cardiol 39: 137-145, 1977.

Kohanna FH, Cunningham JN: Monitoring of cardiac output by thermodilution after open heart surgery. J Thoracic Cardiovas Surg 73:451-457, 1977.

Scheuer-Leeser M, Morgust A, Reul H, Irnich W: Some aspects to the pulsation error in blood flow calculations by indicator dilution techniques. Med Biol Engineer Comput 15:118-123, 1977.

Vandermoten P, Bernard R, de Hemptinne J, et al: Cardiac output monitoring during the acute phase of myocardial infarction: accuracy and precision of the thermodilution method. Cardiology 62:291-295, 1977.

Fischer AP, Benis AM, Jurado RA, et al: Analysis of errors in measurement of cardiac output by simultaneous dye and thermodilution in cardiothoracic surgical patients. Cardiovas Res 12:190-199, 1978.

Hoel BH: Some aspects of the clinical use of thermodilution in measuring cardiac output. Scand J Clin Lab Invest 38:383-388, 1978.

Moodie DS, Feldt RH, Kaye MP, et al: Measurement of cardiac output by thermodilution; development of accurate measurements at flows applicable to the pediatric patient. J Surg Res 25:305-311, 1978.

McMichan JC, Michel L: Knotting of central venous catheters: nonsurgical correction. Chest 74:572-573, 1978.

Powner DJ, Snyder JV: In vitro comparison of six commercially available thermodilution cardiac output systems. Med Instrument 12:122, 1978.

Levett JM, Replogle RL: Thermodilution cardiac output: a critical analysis and review of the literature. J Surg Res 27:392-404, 1979.

Nitzan M, Weinreb A, Appelbaum A: Theoretical evaluation of the thermodilution method for cardiac output determination. IEEE Trans Biomed Engineer BME 27:613-615, 1980.

Runciman WB, Ilsley AH, Roberts JG: Thermodilution cardiac output - a systematic error. Anaesth Intens Care 9:135-139, 1981.

American Edwards Laboratories: Understanding hemodynamic measurements made with the Swan-Ganz catheter. American Edwards Laboratories, Santa Ana, CA, 1982.

Bilfinger TV, Lin CY, Anagnostopoulos CE: In vitro determination of accuracy of cardiac output measurements by thermodilution. J Surg Res 33:409-414, 1982.

Mammana RB, Hiro S, Levitsky S, et al: Inaccuracy of pulmonary capillary wedge pressure when compared to left atrial pressure in the early postsurgical period. J Thoracic Cardiovas Surg 84:420-425, 1982.

Shell WE, DeWood MA, Peter T, et al: Comparison of clinical signs and hemodynamic state in the early hours of transmural myocardial infarction. Am Heart J 104:521-528, 1982.

Snyder JV, Powner DJ: Effects of mechanical ventilation on the measurement of cardiac output by thermodilution. Crit Care Med 10:677-682, 1982.

Boyd KD, Thomas SJ, Gold J, Boyd, AD: A prospective study of complications of pulmonary artery catheterizations in 500 consecutive patients. Chest 84:245-249, 1983.

Connors AF, McCaffre DR, Gray BA: Evaluation of right-heart catheterization in the critically ill patient without acute myocardial infarction. N Engl J Med 308:263-267, 1983.

Elkayam U, Berkley R, Azen S, et al: Cardiac output by thermodilution technique: effect of injectate's volume and temperature on the accuracy and reproducibility in the critically ill patient. Chest 84:418-422, 1983.

Niederberger M, Gaul G: Use of flow-directed catheters for assessment of left ventricular function during exercise; methods, risks, and meaning for cardiac rehabilitation. J Cardiac Rehab 3:780-787, 1983.

Shellock FG, Riedinger MS: Reproducibility and accuracy of using room temperature vs ice temperature injectate for thermodilution cardiac output determination. Heart & Lung 12:175-176, 1983.

Shellock FG, Riedinger MS, Bateman TM, Gray RJ: Thermodilution cardiac output determination in hypothermic surgery patients: room vs. ice temperature injectate. Crit Care Med 11:668-670, 1983.

Swan HJC, Ganz W: Hemodynamic measurements in clinical practice: a decade in review. J Am Coll Cardiol 1:103-113, 1983.

Woog RH, McWilliam DB: A comparison of methods of cardiac output measurement. Anaesth Intens Care 11:141-146, 1983.

Vennix CV, Nelson DH, Pierpont GL: Thermodilution cardiac output in critically ill patients: comparison of room temperature and iced injectate. Heart & Lung 13:574-578, 1984.

Reidinger MS, Shellock FG: Technical aspects of the thermodilution method for measuring cardiac output. Heart & Lung 13:215-221, 1984.

Rowley KM, Clubb KS, Smith GJW, Cabin HS: Right-sided infective endocarditis as a consequence of flow-directed pulmonary artery catheterization. N Engl J Med 311:1152-1156, 1984.

Barcelona M, Patague L, Bunoy M, et al: Cardiac output determination by the thermodilution method: comparison of ice-temperature injectate versus room-temperature injectate contained in prefilled syringes or a closed injectate delivery system. Heart & Lung 14:232-235, 1985.

Conners AF, Castele RJ, Farahat NZ, Tomashefski JF: Complications of right heart catheterization; a prospective autopsy study. Chest 88:567-572, 1985.

Iberti TI, Benjamin E, Gruppi L, Raskin JM: Ventricular arrhythmias during pulmonary artery catheterization in the intensive care unit. Am J Med 78:451-454, 1985.

Latson T, Maruschak G: A faulty lumen resulting in erroneous thermodilution cardiac output measurement. J Clin Monit 1:213-215, 1985.

Kadota LT: Theory and application of thermodilution cardiac output measurement: a review. Heart & Lung 14:605-614, 1985.

Nelson LD, Anderson HB: Patient selection for iced versus room temperature injectate for thermodilution cardiac output determination. Crit Care Med 13:182-184, 1985.

Robin ED: The cult of the Swan-Ganz catheter: overuse and abuse of pulmonary flow catheters. Ann Intern Med 103:445-449, 1985.

Streisand JB, Clark NJ, Pace NL: Pulmonary arterial catheterization before anesthesia in patients undergoing cardiac surgery. J Clin Monit 1:193-196, 1985.

Amin DK, Shah PK, Swan HJC: The Swan-Ganz catheter; choosing and using the equipment. J Crit Illness 1(4):34-37, 1986.

Amin DK, Shah PK, Swan HJC: The Swan-Ganz catheter; insertion technique. J Crit Illness 1(4):38-45, 1986.

Amin DK, Shah PK, Swan HJC: The Swan-Ganz catheter; indications for insertion. J Crit Illness 1(5):54-61, 1986.

Amin DK, Shah PK, Swan HJC: The Swan-Ganz catheter; tips on interpreting results. J Crit Illness 1(5):40-48, 1986.

Desbiens NA: The balloon-tipped thermodilution catheter; aspects of its clinical utility. Postgrad Med 79(6):109-117, 1986.

Lyons K, Dalbow M: Room temperature injectate and iced injectate for cardiac output: a comparative study. Crit Care Nurse 6(1):48-50, 1986.

Nadeau S: Limitations of cardiac output measurements by thermodilution. CN Anaesthesiol Soc J 33:780-784, 1986.

Mackenzie JD, Haites NE, Rawles JM: Method of assessing the reproducibility of blood flow measurement: factors influencing the performance of thermodilution cardiac output computers. Br Heart J 55:14-24, 1986.

Schmitt EA, Brantigan CO: Common artifacts of pulmonary artery and pulmonary artery wedge pressures; recognition and interpretation. J Clin Monit 2:44-52, 1986.

Swan HJC, Shar PK: The rationale for bedside monitoring. J Crit Illness 1(4):24-28, 1986.

Van Hook CJ, Carilli AD, Haponik EF: Hemodynamic effects of positive end expiratory pressure. Am J Med 81:307-310, 1986.

Becker RC, Martin RC, Underwood DA: Right-sided endocardial lesions and flow-directed pulmonary artery catheters. Cleve Clinic J Med 54:384-388, 1987.

Combs DT, Hanlon JT: In vivo comparison of two thermodilution systems. Chest 95:926-928, 1987.

Gardner PE, Monat LA, Woods SL: Accuracy of the closed injectate delivery system in measuring thermodilution cardiac output. Heart & Lung 16:552-561, 1987.

Green LM: Thermodilution cardiac output. J Post Anesthsiol Nurs 2(2):132-137, 1987.

Matthew EB, Vender JS: Comparison of thermodilution cardiac output measured by different computers (letter). Crit Care Med 15:989, 1987.

Raffin TA: The technique of thermodilution cardiac output measurements. J Crit Illness 2(1):73-79, 1987.

Cross JA, Vargo RL: Cardiac output: iced vs room temperature solution. Dimens Crit Care Nurs 7(3):146-149, 1988.

Emergency Care Research Institute (ECRI): Cardiac output units, thermodilution. ECRI Feb; 1988.

Gill JB, Cairns JA: Prospective study of pulmonary artery balloon flotation catheter insertions. J Intens Care Med 3:121-128, 1988.

Goldberg RJ: Risks and benefits of pulmonary artery catheterization. J Intens Care Med 3:69-70, 1988.

Mazzara JT, Parmley WM, Russell RO: A close look at Swan-Ganz catheters. Patient Care Feb 15; 37, 1988.

Yonkman CA, Hamory BH: Sterility and efficiency of two methods of cardiac output determination: closed loop and capped syringe methods. Heart & Lung 17:121-128, 1988.

Bourdillon PDV, Fineberg N: Comparison of iced and room temperature injectate for thermodilution cardiac output. Cathet Cardiovas Diag 17:116-120, 1989.

Rajput MA, Richey HM, Bush BA, et al: A comparison between a conventional and a fiberoptic flow-directed thermodilution pulmonary artery catheter in critically ill patients. Arch Intern Med 149:83-85, 1989.

Smart FW, Husserl FE: Complications of flow-directed balloon-tipped catheters. Chest 97:227-228, 1990.

11.2 Cardiac Output Measurement by Electrical Bioimpedance

Ramos MU: An abnormal early diastolic impedance waveform: a predictor of poor prognosis in the cardiac patient? Am Heart J 94:274-281, 1977.

Boer P, Roos JC, Geyskes GG, Dorhout Mees EJ: Measurement of cardiac output by impedance cardiography under various conditions. Am J Physiol 237(H491-H496), 1979.

Sakamoto K, Muto K, Kanai H, Iizuka M: Problems of impedance cardiography. Med Biol Engineers Comput 17:697-709, 1979.

Rubal BJ, Baker LE, Poder TC: Correlation between maximum dZ/dt and parameters of left ventricular performance. Med Biol Engineers Comput 18:541-548, 1980.

Djordjevich L, Sadove MS: Experimental study of the relationship between the base impedance and its time derivative in impedance plethysmography. Med Physiol 8:76-78, 1981.

Miles DS, Sawka MN, Wilde SW, et al: Estimation of cardiac output by electrical impedance during arm exercise in women. J Appl Physiol 51:1488-1492, 1981.

Schieken RM, Patel MR, Falsettl HL, Lauer RM: Effect of mitral regurgitation on transthoracic impedance cardiogram. Br Heart J 45:166-172, 1981.

Bo Sramek B: Cardiac output by electrical impedance. Med Electron April:93-97, 1982.

Bo Sramek B: Bomed's electrical bioimpedance technology for thoracic applications. Bomed, 1985.

Parulkar GB, Jindal GD, Bhardwaj R, et al: Impedance cardiography in mitral valve diseases. Indian Heart J 37:37-42, 1985.

Patterson RP: Sources of the thoracic cardiogenic electrical impedance signal as determined by a model. Med Biol Engineer Comput 23:411-417, 1985.

Appel PL, Kram HB, Mackabee J, et al: Comparison of measurements of cardiac output by bioimpedance and thermodilution in severely ill surgical patients. Crit Care Med 14:933-935, 1986.

Bernstein DP: Continuous noninvasive real-time monitoring of stroke volume and cardiac output by thoracic electrical bioimpedance. Crit Care Med 14:898-901, 1986.

Bernstein DP: A new stroke volume equation for thoracic electrical bioimpedance: theory and rationale. Crit Care Med 14:904-909, 1986.

Donovan KD, Dobb GJ, Woods PD, Hockings BE: Comparison of transthoracic electrical impedance and thermodilution methods for measuring cardiac output. Crit Care Med 14:1038-1044, 1986.

Wagner J, Geddes LA, Foster K, Farag A: Monitoring heart and respiratory activity by impedance change using neck electrodes. Med Biol Engineer Comp 25:100-102, 1987.

Smith SA, et al: Automated non-invasive measurement of cardiac output: comparison of electrical bioimpedance and carbon dioxide rebreathing techniques. Br Heart J 59:292-298, 1988.

Miles DS, Gotshall RW, Golden JC, et al: Accuracy of electrical impedance cardiography for measuring cardiac output in children with congenital heart defects. Am J Cardiol 61:612-616, 1988.

Spinale FG, Reines HD, Crawford FA: Comparison of bioimpedance and thermodilution methods for determining cardiac output: experimental and clinical studies. Ann Thorac Surg 45:421-425, 1988.

Salandin V, Zussa C, Risica G, et al: Comparison of cardiac output estimation by thoracic electrical bioimpedance, thermodilution, and Fick methods. Crit Care Med 16:1157-1158, 1988.

11.3 Cardiac Output Measurement by Doppler Echocardiography

Goldberg SJ, Sahn DJ, Allen HD, et al: Evaluation of pulmonary and systemic blood flow by 2-dimensional Doppler echocardiography using fast Fourier transform spectral analysis. Am J Cardiol 50:1394-1400, 1982.

Huntsman LL, Stewart DK, Barnes SR, et al: Noninvasive Doppler determination of cardiac output in man: clinical validation. Circulation 67:593-602, 1983.

Chandraratna PA, Nanna M, McKay C, et al: Determination of cardiac output by transcutaneous continuous-wave ultrasonic Doppler computer. Am J Cardiol 53:234-237, 1984.

Gardin JM, Burn CS, Childs WJ, Henry WL: Evaluation of blood flow velocity in the ascending aorta and main pulmonary artery of normal subjects by Doppler echocardiography. Am Heart J 107:310-319, 1984.

Nishimura RA, Callahan MJ, Schaff HV, et al: Noninvasive measurement of cardiac output by continuous-wave Doppler echocardiography: initial experience and review of the literature. Mayo Clinic Proc 59:484-489, 1984.

Schuster AH, Nanda N: Doppler echocardiographic measurement of cardiac output; comparison with a non-golden standard. Am J Cardiol 53:257-259, 1984.

Wilson N, Goldberg SJ, Dickinson DF, Scott O: Normal intracardiac and great artery blood velocity measurements by pulsed Doppler echocardiography. Br Heart J 53:451-458, 1985.

Louie EK, Maron BJ, Green KJ: Variations in flow-velocity waveforms obtained by pulsed Doppler echocardiography in the normal human aorta. Am J Cardiol 58:821-826, 1986.

McLennan FM, Haites NE, Mackenzie JK, et al: Reproducibility of linear cardiac output measurement by Doppler ultrasound alone. Br Heart J 55:25-31, 1986.

Wallmeyer K, Wann LS, Sagar KB, et al: The influence of preload and heart rate on Doppler echocardiographic indexes of left ventricular performance: comparison with invasive indexes in an experimental preparation. Circulation 74:181-186, 1986.

Bouchard A, Blumein S, Schiller NB, et al: Measurement of left ventricular stroke volume using continuous wave Doppler echocardiography of the ascending aorta and M-mode echocardiography of the aortic valve. J Am Coll Cardiol 9:75-83, 1987.

Daley PJ, Sagar KB, Collier BD, et al: Detection of exercise induced changes in left ventricular performance by Doppler echocardiography. Br Heart J 58:447-454, 1987.

Davies GG: Comparison of continuous esophageal Doppler cardiac output with continuous Fick cardiac output during hemorrhage in pigs (abstract). J Clin Monit 3:296, 1987.

Donovan KD, Dobb GJ, Newman MA, et al: Comparison of pulsed Doppler and thermodilution methods for measuring cardiac output in critically ill patients. Crit Care Med 15:853-857, 1987.

Ihlen H, Endressen, Golf S, Nitter-Hauge S: Cardiac stroke volume during exercise measured by Doppler echocardiography: comparison with the thermodilution technique and evaluation of reproducibility. Br Heart J 58:455-459, 1987.

Gardin JM, Sung H, Yognathian AP, et al: Doppler flow velocity mapping in an in vitro model of the normal pulmonary artery. J Am Coll Cardiol 12:1366-1376, 1988.

Griffith MJ, Mehta D, Ward DE, Camm AJ: Ascending aorta Doppler echocardiography in the diagnosis of broad complex tachycardia. Am Heart J 116:555-557, 1988.

Harrison MR, Berk MR, DeMaria AN: Exercise Doppler study of cardiac function. Cardiovas Rev & Rep Feb; 33-38, 1988.

Johnson GL, Moffet CB, Noonan JA: Doppler echocardiographic studies of diastolic ventricular filling patterns in premature infants. Am Heart J 116:1568-1574, 1988.

Mathias DW, Wann LS, Sagar KB, Klopfenstein HS: The effect of regional myocardial ischemia on Doppler echocardiographic indexes of left ventricular performance: influence of heart rate, aortic blood pressure, and the size of the ischemic zone. Am Heart J 166:953-960, 1988.

Robson S, Murray A, Peart I, et al: Reproducibility of cardiac output measurement by cross-sectional and Doppler echocardiography. Br Heart J 59:680-684, 1988.

Yoganathian AP, Cape EG, Sung H, et al: Review of hydrodynamic principles for the cardiologist: applications to the study of blood flow and jets by imaging techniques. J Am Coll Cardiol 12:1344-1353, 1988.

Aguirre FV, Pearson AC, Lewen MK, et al: Usefulness of Doppler echocardiography in the diagnosis of congestive heart failure. Am J Cardiol 63:1098-1102, 1989.

Burstow DJ, Oh JK, Bailey KR, et al: Cardiac tamponade: characteristic Doppler observations. Mayo Clinic Proc 64:312-324, 1989.

Come PC: Echo diagnosis of pulmonary hypertension. Cardiology March 136-143, 1989.

Cyran SE, Kimball TR, Meyer RA, et al: Efficacy of interoperative transesophageal echocardiography in children with congenital heart disease. Am J Cardiol 63:594-598, 1989.

Desruennes M, Cabrol C: Acute cardiac allograft rejection: detection by Doppler echocardiography. Cardiol Board Rev 6:55-63, 1989.

Devereaux RB: Left ventricular diastolic dysfunction: early diastolic relaxation and late diastolic compliance (editorial). J Am Coll Cardiol 13:337-339, 1989.

Ferguson JJ, Bush HS, Riuli EP: Doppler echocardiographic assessment of the effect of balloon aortic valvuloplasty on left ventricular systolic function. Am Heart J 117:18-24, 1989.

Ferguson JJ, Manning WJ, Come PC: Pulsed Doppler echocardiographic determination of the time course of left ventricular filling: validation with cineangiography. Am Heart J 117:127-132, 1989.

Feigenbaum H: Echocardiographic evaluation of left ventricular diastolic function (editorial). J Am Coll Cardiol 13:1027-1029, 1989.

Fisher EA, Stahl JA, Goldman ME: Color-flow echocardiography: aortic insufficiency. Primary Cardiol March; 78-83, 1989.

Fisher EA, Stahl JA, Ritter SB, Goldman ME: Clinical applications of transesophageal echocardiography. Primary Cardiol July; 53-56, 1989.

Harrison MR, Clifton D, Sublett KL, DeMaria AN: Effect of heart rate on Doppler indexes of systolic function in humans. J Am Coll Cardiol 14:929-935, 1989.

Isaaz, K, Ethevenot G, Admant P, et al: A new Doppler method of assessing left ventricular ejection force in chronic congestive heart failure. Am J Cardiol 64:81-87, 1989.

Klein AL, Hatle LK, Burstow DJ, et al: Doppler characterization of left ventricular diastolic function in cardiac amyloidosis. J Am Coll Cardiol 13:1017-1026, 1989.

Kumar A, Minagoe S, Thangathurai D, et al: Noninvasive measurement of cardiac output during surgery using a new continuous-wave Doppler esophageal probe. Am J Cardiol 64:793-798, 1989.

Looyenga DS, Liebson PR, Bone RC, et al: Determination of cardiac output in critically ill patients by dual beam Doppler echocardiography. J Am Coll Cardiol 13:340-347, 1989.

Maeda M, Yokota M, Iwase M, et al: Accuracy of cardiac output measured by continuous wave Doppler echocardiography during dynamic exercise testing in the supine position in patients with coronary artery disease. J Am Coll Cardiol 13:76-83, 1989.

Maze SS, Kottler MN, Parry MN: Doppler evaluation of changing cardiac dynamics during Cheyne-Stokes respiration. Chest 95:525-529, 1989.

Morera J, Hoadley SD, Roland JM, et al: Estimation of the ratio of pulmonary to systemic pressures by pulsed-wave Doppler echocardiography for assessment of pulmonary arterial pressures. Am J Cardiol 63:862-866, 1989.

Nishimura RA, Housmans PR, Hatle LK, et al: Assessment of diastolic function of the heart: background and current applications of Doppler echocardiography. Part I. Physiologic and pathophysiologic features. Mayo Clinic Proc 64:71-81, 1989.

Nishimura RA, Abel MD, Hatle LK, Tajik AJ: Assessment of diastolic function of the heart: background and current applications of Doppler echocardiography. Part II. Clinical Studies. Mayo Clinic Proc 64:181-204, 1989.

Nishimura RA: Another measurement of cardiac output: is it truly needed? (editorial). J Am Coll Cardiol 13:1393-1394, 1989.

Roman MJ, Devereaux RB, Kramer-Fox R, et al: Two-dimensional echocardiographic aortic root dimensions in normal children and adults. Am J Cardiol 64:507-512, 1989.

Segal J, Pearl RG, Ford AJ, et al: Instantaneous and continuous cardiac output obtained with a Doppler pulmonary artery catheter. J Am Coll Cardiol 13:1382-1392, 1989.

Simpson IA, Valdes-Cruz LM, Sahn DJ, et al: Doppler color flow mapping of simulated in vitro regurgitant jets: evaluation of the effects of orifice size and hemodynamic variables. J Am Coll Cardiol 13:1197-1207, 1989.

Spodick DH, Koito H: Nongeometric Doppler stroke volume determination. Am J Cardiol 63:883-884, 1989.

Stoddard MF, Pearson AC, Kern MJ, et al: Left ventricular diastolic function: comparison of pulsed Doppler echocardiographic and hemodynamic indexes in subjects with and without coronary artery disease. J Am Coll Cardiol 13:327-336, 1989.

Stoddard MF, Chaitman BR, Byers SL, et al: Noninvasive assessment of diastolic and systolic properties of ibopamine in patients with congestive heart failure. Am Heart J 117:395-402, 1989.

Stoddard MF, Pearson AC, Kern MJ, et al: Influence of alteration of preload on the pattern of left ventricular diastolic filling as assessed by Doppler echocardiography in humans. Circulation 79:1226-1236, 1989.

Oh JK, Khandheria BK, Seward JB, Tajik AJ: Transesophageal echocardiography: anatomic orientations, instrumentation, and indications. Cardiovas Rev & Rep June; 22-29, 1989.

11.4 Noninvasive Measurement of Cardiac Output: Miscellaneous Techniques

Cerritelli P, Fahri LE: Readjustments in cardiac output and gas exchange during onset of exercise and recovery. J Appl Physiol 21:1345-1350, 1966.

Kim TS, Rahn H, Farhi LE: Estimation of true venous and arterial PcO_2 by gas analysis of a single breath. J Appl Physiol 21:1338-1344, 1966.

Gilbert R, Auchincloss JH: Comparison of single-breath and indicator dilution measurement of cardiac output. J Appl Physiol 29:119-122, 1970.

Hlastala MP, Wranne B, Lenfant CJ: Single-breath method of measuring cardiac output: a re-evaluation. J Appl Physiol 33:846-848, 1972.

Chen H, Silverton NP, Hainsworth R: Evaluation of a method for estimating cardiac output from a single breath in humans. J Appl Physiol 53:1034-1038, 1982.

Haites NE, McLennan FM, Mowat DHR, Rawles JM: How far is the cardiac output? Lancet 2:1025-1027, 1984.

Tremper KK: Continuous noninvasive cardiac output: Are we getting there? Crit Care Med 15:278-279, 1987.

Gurry MK, Freedson PS, Kline G, et al: A comparative analysis of an automated noninvasive estimate of cardiac output with direct Fick and thermodilution techniques. J Cardiopulmonary Rehab 9:122-126, 1989.

Lange RA, Dehmer GJ, Wells PJ, et al: Limitations of a metabolic rate meter for measuring oxygen consumption and cardiac output. Am J Cardiol 64:783-786, 1989.

12.0 GLOSSARY

Afterload—The resistance against which the left ventricle of the heart ejects blood.

Aorta—The main artery that receives the output of the left ventricle of the heart and distributes blood to the entire body.

Artifact—Distortion, aberration or inaccuracy in the data obtained from a monitoring device.

Autonomic nervous system—The division of the nervous system that regulates involuntary action, such as that of the heart, intestines, and glands; divided into the sympathetic and parasympathetic nervous systems.

Bioimpedance—Technique for measuring cardiac output that makes use of the change in electrical resistance of the chest occurring with ejection of blood from the left ventricle of the heart into the aorta.

Blood gas analyzer —A laboratory instrument dedicated to the measurement of pH and partial pressures of oxygen and carbon dioxide in blood.

Calorie deficit—The difference in heat content between blood and the cold indicator fluid that is injected for the determination of cardiac output by thermodilution.

Cardiac index—Cardiac output divided by the body surface area; $l/min/m^2$.

Cardiac output—The volume of blood pumped by the heart per unit time, usually expressed in liters per minute (l/min); also the product of heart rate and stroke volume.

Cardiovascular system—The heart and blood vessels.

Carrier frequency—The frequency of sound emitted by the sending unit of a Doppler ultrasound device.

Catheter—A tubular medical device for insertion into the body in order to withdraw fluids, keep a passage open, or measure an internal body parameter such as a pressure.

Central venous pressure (CVP)—The venous pressure as measured at the right atrium; also called right atrial pressure (RAP).

Circulatory system—See cardiovascular system.

Compliance—The change in volume of a hollow, distensible structure resulting from the application of a pressure differential across the wall of the structure; specifically, the relationship between pressure and volume in the left ventricle of the heart.

Contractility—The ability of a muscle to shorten or develop increased tension.

Co-oximeter—An instrument for measuring the degree that hemoglobin in blood is combined with oxygen, usually expressed as the percentage saturation.

Densitometer—An instrument for measuring the optical density of a material.

Dextrose solution—A liquid mixture of water and dextrose, a sugar (also called glucose).

Diastole—The portion of the cardiac cycle when the heart fills with blood, prior to systole.

Doppler principle—An apparent change in the frequency of waves, as of sound or light, occurring when the source and observer are in motion relative to one another, with the frequency increasing when the source and observer approach one another and decreasing when they move apart.

Dye dilution method—An indicator dilution method for measuring cardiac output that uses dye, usually indocyanine green, as the indicator substance.

Endotracheal tube—A tube inserted into the trachea to establish a clear passage for breathing.

Fick method—A standard laboratory method for determining cardiac output by measuring the rate of oxygen uptake from the lungs and the oxygen content of arterial and central venous blood.

Fluoroscopy—An x-ray device that projects the radiographic images onto a video screen, rather than onto photographic film, in order that the images can be viewed in real time.

Frank-Starling mechanism—The relationship between the stroke work and the end-diastolic volume of the left ventricle of the heart. In clinical practice, stroke volume or cardiac output are often substituted for stroke work, and end-diastolic volume is replaced by pulmonary artery wedge pressure (end-diastolic volume refers to the volume of blood in the ventricle at the moment immediately preceding systole, also the largest volume of the ventricle during the cardiac cycle).

Heart rate—The number of beats (contractions) of the heart in a unit of time (usually beats per minute).

Hemoglobin—The iron-containing protein in red blood cells that carries oxygen.

Hormone—A chemical messenger produced by one organ and then carried in the blood stream to another organ that is chemically stimulated by the substance.

Hypertonic saline—A solution of salt and water that has an osmolarity greater than blood.

Hypothermia—Body temperature that is below normal.

Indicator dilution technique—A method for measuring cardiac output. A substance is injected into the circulation and the concentration of this substance is measured downstream from the injection site. The extent of dilution of the indicator is directly proportional to cardiac output.

Inotrope—Drug that increases cardiac contractility.

Mean arterial pressure—The mean blood pressure in the arteries.

Mixed venous oxygen saturation (of hemoglobin)—Abbreviated as $S\bar{v}O_2$, a measurement of the oxygen saturation of hemoglobin at the point of oxygen for carbon dioxide exchange; reflects the balance between oxygen delivery and oxygen consumption in the body as a whole.

Nomogram—A chart representing numerical relationships; also called a nomogram or alignment chart.

Partial pressure of oxygen (PO_2)—The pressure that oxygen would exert if it were alone in a container.

Partial pressure of arterial oxygen (PaO_2)—Partial pressure of oxygen in arterial blood.

Partial pressure of venous oxygen (PvO_2)—Partial pressure of oxygen in venous blood, usually venous blood from the pulmonary artery (also called mixed venous blood).

Pneumothorax—The accumulation of air in the pleural cavity because of a hole in the lung or chest wall.

Preload—In isolated heart muscle, the force used to stretch the muscle to a particular length. In the intact heart, the end-diastolic wall stress. Because end-diastolic wall stress is not easily determined, end-diastolic pressure or volume are usually used in the clinical setting to estimate preload.

Pulmonic valve—The heart valve between the right ventricle and the pulmonary artery.

Pulmonary artery catheter—A catheter designed to be inserted in the pulmonary artery. The most commonly used type of pulmonary artery catheter, balloon-tipped and flow-directed type, is also called the Swan-Ganz catheter.

Pulmonary artery wedge pressure—The downstream pressure measured from the distal port of the pulmonary artery catheter with the balloon inflated to occlude the pulmonary artery. This pressure usually reflects left atrial and left ventricular end-diastolic pressures; also called the pulmonary artery occlusion pressure or pulmonary capillary wedge pressure.

Spectrophotometry—A laboratory technique for measuring the alteration of light passed through a substance in order to characterize or quantify the substance.

Sternal notch—The indentation of the upper edge of the sternum (breast bone).

Stethoscope—An instrument used to listen to sounds produced within the body.

Stroke volume—The amount of blood ejected by each cycle of contraction of the heart.

Swan-Ganz catheter—See pulmonary artery catheter.

Systole—Contraction of the heart muscle.

Thermistor—A resistor made of semiconductors having resistance that varies rapidly and predictably with temperature.

Thermodilution—An indicator dilution technique for measuring cardiac output that uses a cold fluid as the indicator substance.

Theta—For a Doppler ultrasound device, the angle between the emitted sound and the moving object that reflects the sound.

Tricuspid valve—The heart valve between the right atrium and right ventricle.

Ultrasound—Ultrahigh frequency sound (20,000 cycles per second).

Ventilatory cycle—Alternating inflation and deflation of the lungs.

Wedge pressure—See pulmonary artery wedge pressure.

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